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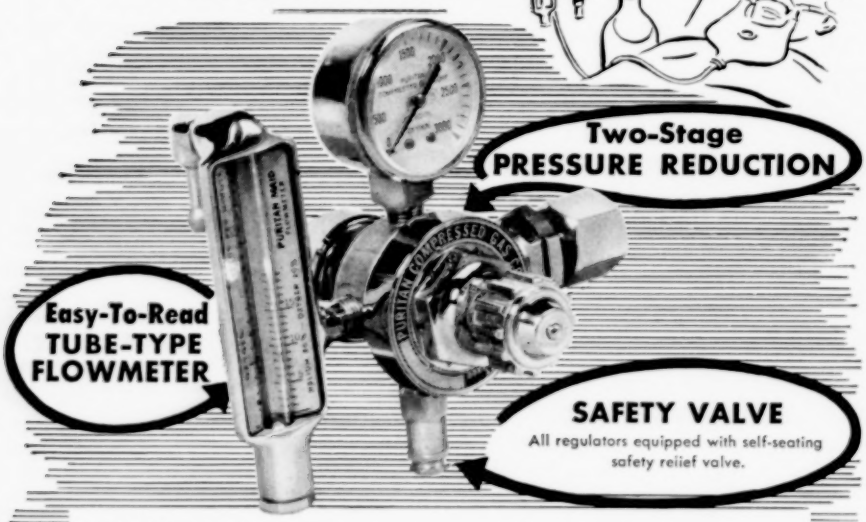

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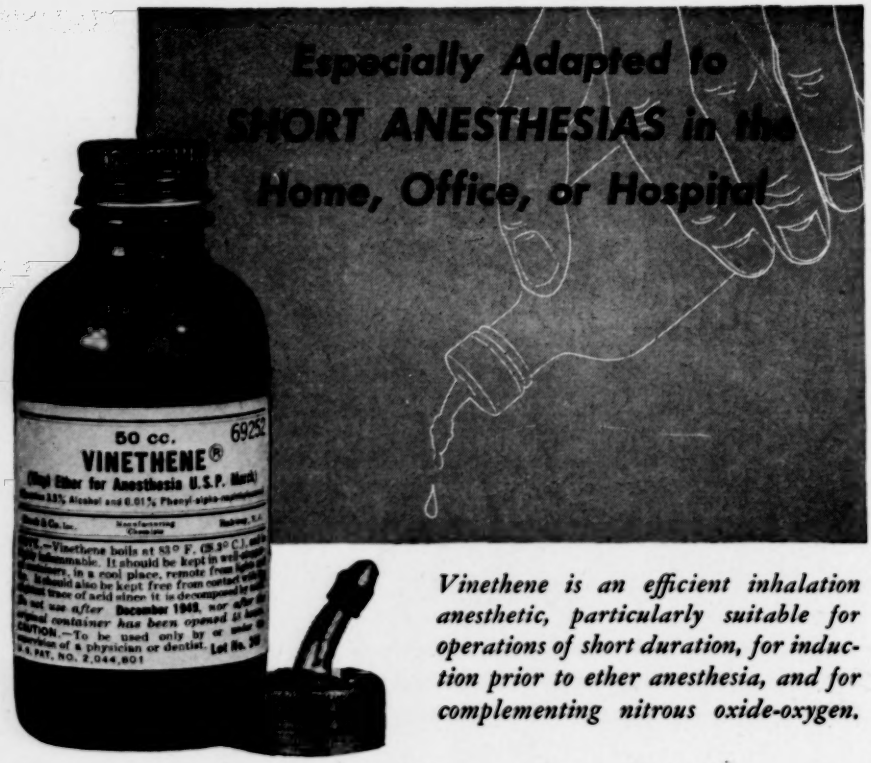

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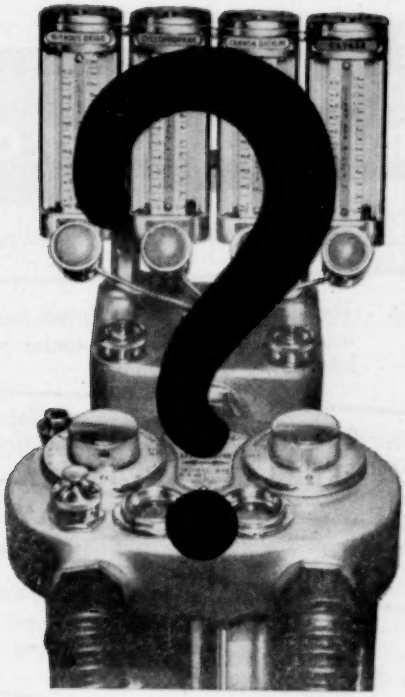

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The Journal of the American Association of Nurse Anesthetists

VOLUME XVI NOVEMBER, 1948 NUMBER FOUR

ALTERED PHYSIOLOGY OF NORMAL BODY FUNCTIONS DURING ANESTHESIA

Esther M. Greisheimer, Ph.D., M.D.*
Philadelphia

Introduction

Whenever an anesthetic is administered, changes occur in practically every system of the body, and the means of compensating for adverse conditions are greatly curtailed; therefore the anesthetist must do all in her power to make conditions as favorable as possible for the patient.

I shall endeavor to point out the principal changes which occur in the nervous system, respiratory system, circulatory system, gastrointestinal system, and renal division of the excretory system.

Nervous System

The functions of the various portions of the central nervous system will be listed. The functions of the cerebral hemispheres are consciousness, sensation, voluntary motion, and regulation of the activities of the lower levels of the nervous system. The thalamus is the center in which crude sensations are appreciated, and in which sensory impulses are sorted out and relayed on to the cerebral cortex. The basal ganglia control the stereotyped movements which normally accompany voluntary movements, such as crossing the legs when sitting down, swinging the arms when walking, and gesticulating when talking. The hypothalamus controls the activities of the autonomic system. The medulla contains the vital centers which control respiration, heart, and blood pressure. The spinal cord conducts impulses to and from the brain and serves as a reflex center.

During anesthesia many changes take place in the nervous system. In stage one, as shown in figure 1a, there is clouding of consciousness; the patient loses the power of judgment, is disoriented, and cannot interpret impulses which reach the cerebrum. All cerebral functions are depressed, but the reflexes whose centers lie at lower levels of the central nervous system are intact. The respiration may be fast and irregular or periods of apnea may be noted. Anesthetics impair oxidative processes, especially in the gray matter of the central nervous system, and the activities of the cerebral cortex are curtailed. The manner in which the change in oxidation is brought about is still a contro-

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*Department of Physiology, Temple University School of Medicine. Illustrated by Dorothy Ellis, A.B.

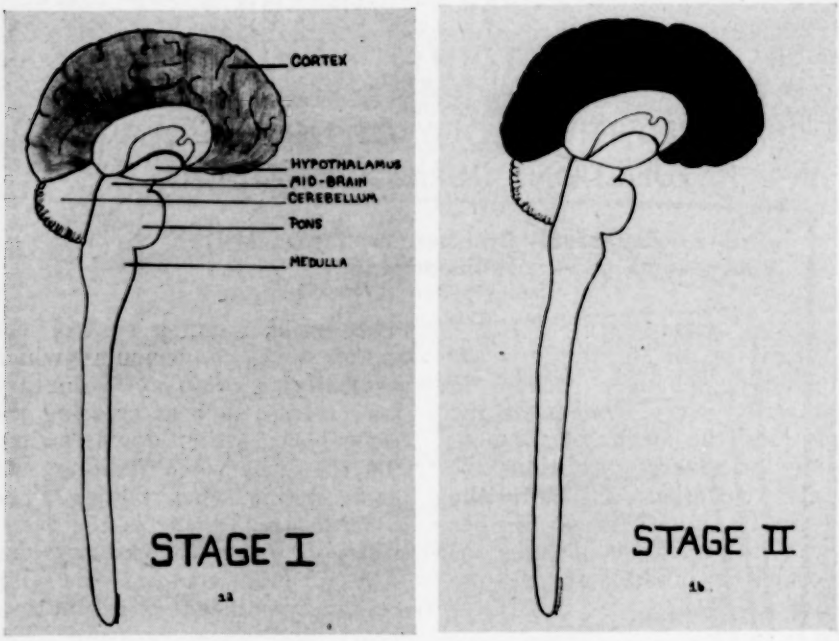


Fig. 1a.—Clouding of consciousness as the anesthetic affects the cerebral cortex.

Fig. 1b.—Loss of consciousness; the subcortical centers are not yet influenced by the anesthetic.

versial question; it is possible that certain anesthetics depress the action of dehydrogenases, which in turn prevents the reduction of cytochrome and thus breaks the link in the chain of oxygen usage by the cells.

In stage two, as shown in figure 1b, the cerebral cortex is still further depressed. As loss of consciousness supervenes, there is hypersensitivity to incoming impulses, which are stopped now at the thalamic level. The thalamus discharges to the basal ganglia and other subcortical levels, and, since the regulating influence of the cerebral cortex is removed, impulses seem to run riot in the lower portions of the central nervous system. This represents the stage of delirium or excitement, in which the responses to stimuli are greatly exaggerated. Painful procedures become dangerous; the beginning of the preparation of the patient may precipitate violent reactions. Reflexes are present and uninhibited; patients may even get off the table if this stage is prolonged. The jaws are set, and muscular tonus is marked. Intubation is impossible. Swallowing, retching, and vomiting are frequent. The respiration is irregular, and respiratory reflexes are exaggerated. The uneven rate of absorption of an inhala-

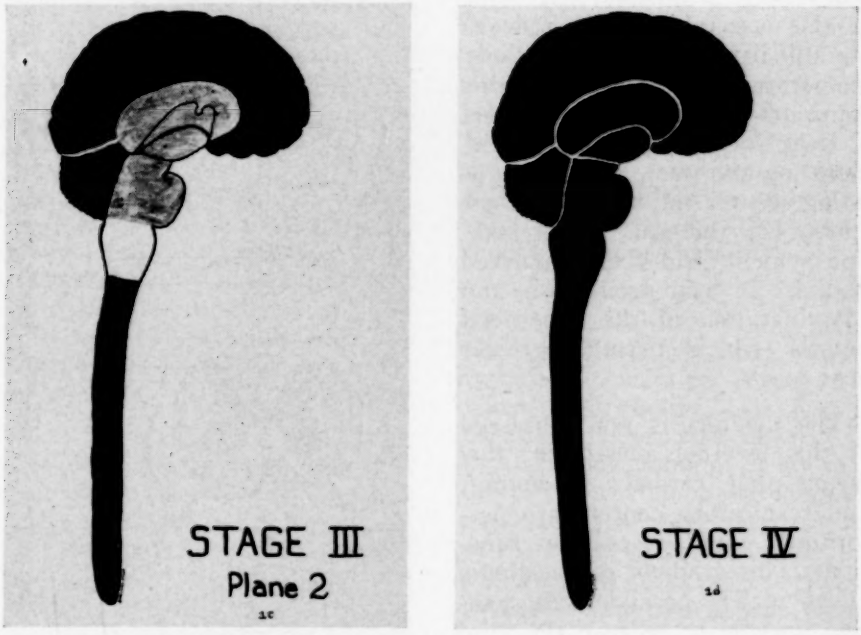


Fig. 1c.—The subcortical centers are under the influence of the anesthetic. The medulla is resistant. The spinal cord is depressed, and many of its reflexes are abolished.

Fig. 1d.—The medulla has come under the influence of the anesthetic; circulation and respiration fail.

tion anesthetic into the blood stream proves to be a difficulty; during apnea the anesthetic reaches low concentrations in the blood stream, while during the deep and rapid breathing it reaches sudden high concentrations, and an even rate of administration is thus made impossible.

At this time, owing to discharges from the hypothalamus, epinephrine is poured into the blood stream in greater than normal amounts, and hypertension occurs. Ventricular fibrillation can occur when epinephrine reaches a heart which has been sensitized by an anesthetic; this is undoubtedly the cause of deaths which occur in stage two. One desires to get the patient through this dangerous second stage as quickly as possible, and the quick acting intravenous anesthetics are deeply appreciated since they shorten this period.

As the patient is carried along to stage three, plane 2, the activities of the thalamus, basal ganglia, hypothalamus, and spinal cord are depressed. The last is especially important since it implies muscular relaxation. The depression of these various portions of the nervous system is indicated in figure 1c. Many

reflexes are abolished, but some of the visceral traction reflexes are still present in plane 2. Body temperature begins to fall at this depth of anesthesia; this is due to three factors: (1) the impairment of the temperature regulating center in the hypothalamus, (2) the muscular relaxation which results in a marked decrease in heat production, and (3) dilatation of the superficial vessels which permits greater heat loss.

The medulla is not depressed at this level of anesthesia; this means that cardiac, vasomotor, and respiratory centers are performing their respective functions. This gradient of anesthetic effect makes possible safe general anesthesia because the vital centers of the medulla are temporarily spared.

As the depth of anesthesia deepens, even the resistant medulla is depressed, as is illustrated in figure 1d, in which stage four is depicted. Respiration and circulation fail, and death results.

Since the aim of the anesthetist is to produce a degree of muscular relaxation satisfactory to the surgeon, the fundamental basis of such relaxation is indicated in figure 2. In the upper diagram the pathway of the normal stretch reflex is shown. A slight stretch of a muscle or pull on a tendon causes a reflex contraction in the muscle stimulated; the reflex is a local one. This reflex contraction of muscles in response to stretch is the basis of muscle tone. The

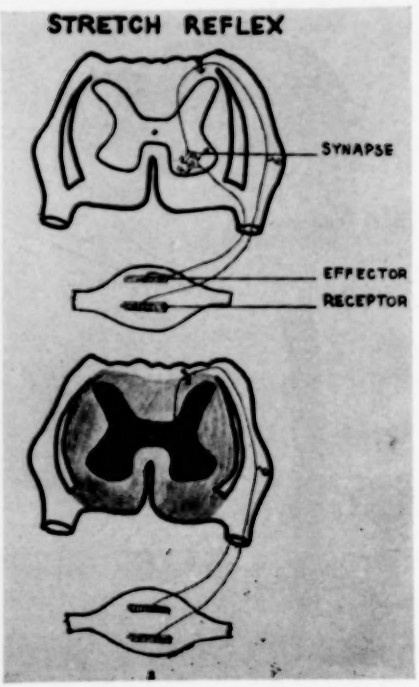


Fig. 2.—The upper diagram shows the pathway of the stretch reflex. The lower diagram shows the loss of the stretch reflex as the spinal cord comes under the influence of the anesthetic.

striated muscles are so arranged on the bony skeleton that a slight stretch is maintained normally. In stage three, plane 2, when the spinal cord is brought under the influence of the anesthetic, the impulses no longer pass through the reflex center, and the stretch reflex is abolished. The abolition of the stretch reflex means muscular relaxation; this is indicated in the lower diagram.

There are some results from muscular relaxation which affect the patient in other ways. As stated before, the decrease in

tone or activity in the striated muscles is responsible to a considerable extent for the drop in body temperature. A second result of muscular relaxation is noted on the circulation, in that the muscular pump mechanism which is so important in the return of venous blood to the heart is now inactive, and the blood stagnates in the venules and veins of the muscles instead of being moved on promptly to the heart. This is one of the factors which induce low blood pressure under anesthesia.

It seems appropriate at this time to mention the fact that this type of muscular relaxation differs markedly from that induced by curare. In the latter case, the block comes at the end of the motor nerve in the vicinity of the effector by preventing the action of acetylcholine on the muscle. Thus, the muscular relaxation produced by curare is not dependent upon depression in the central nervous system and may be produced without any anesthetic.

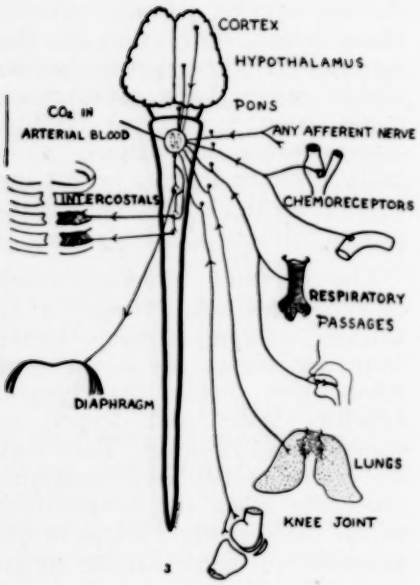
RESPIRATION

Respiration is constantly in the thoughts of the anesthetist, not only when the patient is permitted to do his own breathing, but also when the anesthetist performs controlled or compensated artificial respiration. The respiratory center, which is located in the medulla, is played upon by many factors and is subject to both chemical and nervous control. Figure 3 illustrates the numerous factors which influence the medullary center. The chemical control of the respiratory center is due to the carbon dioxide tension of the arterial blood which flows through the medulla. It is the carbon dioxide which really keeps respiration going from minute to minute, under normal conditions, while the nervous influences adjust the respiration to changes both within and without the body.

The nervous influences come to the medulla from many sources. Impulses may arrive from the cortex, as is the case when one holds his breath, breathes faster and deeper, or thinks about running. They may arrive from the hypothalamus, especially when the temperature of the blood is elevated as in the presence of fever; these hypothalamic impulses appear to be more important in the everyday life of the dog than of man and are responsible for panting in the former.

Impulses may arrive from any receptor of the body and affect the activity of the center; one gasps on seeing a child fall, on hearing a shriek behind him, on stepping unexpectedly into a puddle of cold water, and so on.

The chemoreceptors in the carotid and aortic bodies are especially important in the control of respiration under adverse circumstances; they are the most rugged receptors possessed, with respect to respiration, and may keep respiration going when all other drives fail. These receptors are stimulated when the oxygen tension of the arterial blood is lowered, and thus combine a chemical drive with a reflex control.



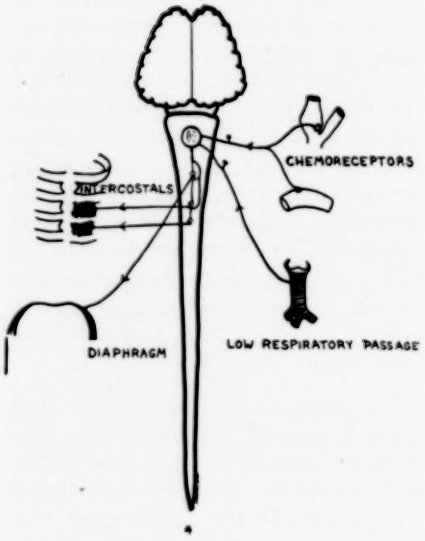


Fig. 3.—Afferent pathways of the impulses which affect the respiratory center in the medulla; it is also affected by carbon dioxide tension of the arterial blood. The efferent pathways to the diaphragm and external intercostal muscles are shown.

Fig. 4.—Loss of many of the respiratory reflexes; only those which arise in the chemoreceptors and the lower respiratory passages remain.

Impulses which arise from the nasal mucous membrane precipitate a sneeze; those which arise from the lower respiratory passages result in a cough.

With each breath one takes, impulses are set up by stretching the alveolar ducts in the lungs; these impulses reach the respiratory center and inhibit it, thus bringing inspiration to a close. This is called the Hering-Breuer reflex, and it normally keeps respiration going at a faster rate than would be the case if the impulses were prevented from reaching the center by section of the vagus nerves. The Hering-Breuer reflex is called a "self-steering" mechanism and is exceedingly important in respiratory control.

During muscular exercise impulses arise from receptors around the joints which speed up respiration and increase the intake of oxygen and output of carbon dioxide; the increased respiratory exchange attained is remarkable.

In spite of the fact that chemical and widespread nervous influences affect the activity of the respiratory center, the effer-

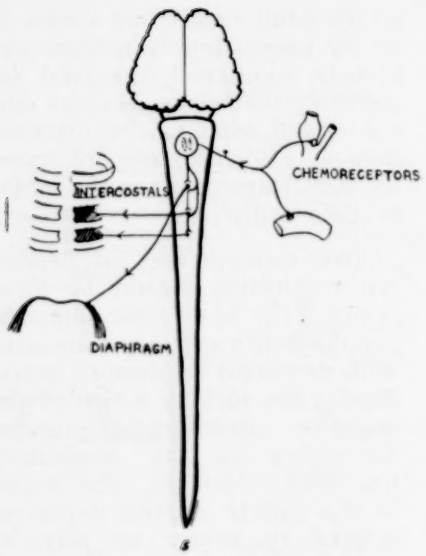
ent pathways for all the reflexes include the phrenic nerves to the diaphragm and the intercostal nerves to the external intercostal muscles principally, as indicated in figure 3. The nerves and muscles which are used in addition during dyspnea are not included.

When ether is given by the open drop method, the respiration is thought to be driven by impulses from the lower respiratory passages. The vapor is so irritating to the mucosa that the tendency to cough is evident with each expiration, which now is forced instead of being passive. Many other reflex effects are absent, as indicated in figure 4.

It must be kept in mind that anesthetics in general, and such premedication agents as morphine, depress the respiratory center. When the center is depressed, it no longer responds to the same concentration of carbon dioxide in the blood as is effective for the normal center. The anesthetist is tempted to increase the carbon dioxide in the anesthetic mixture in order to stimulate respiration; this is dangerous since the high concentration of carbon dioxide exerts a narcotic effect. The hypoxia which characterizes many types of anesthesia, which results from the impaired circulation to the brain or which results from the decreased respiratory movements, likewise takes its toll of the respiratory center. I cannot stress too strongly that the respiratory center is depressed by many factors under anesthesia. It seems fitting at this time to call attention to the fact that sensitive as the adult respiratory center is to the depressing influences previously mentioned, the fetal respiratory center is even more sensitive, and asphyxia neonatorum may be explained in many cases by the treatment given the mother before delivery.

Two consequences of depressed respiration should be mentioned. The first is the impaired venous return which is associated with decreased respiratory movements; the sucking action of the negative intrathoracic pressure diminishes as the respiratory movements decrease. The second is the failure of the depressed respiratory center to play its usual role in the immediate adjustment of the acid-base condition under the changing metabolic levels of anesthesia; this appears as a tendency to acidosis in certain types of anesthesia.

As depression of the respiratory center continues, a point is reached at which respiration is being driven by impulses from the chemoreceptors. This is indicated in figure 5. I wish to remind you that the same low oxygen tension which stimulates the chemoreceptors so effectively at the same time depresses the respiratory center still more. As depression continues, the intercostal muscles become paralyzed, and the diaphragm alone responds to impulses from the respiratory center. This is illustrated in figure 6, which is characteristic of stage three, plane 4. This is called paradoxical respiration; it is dangerous, since ventilation is insufficient. Paradoxical respiration may occur in the deep stages of any type of inhal-



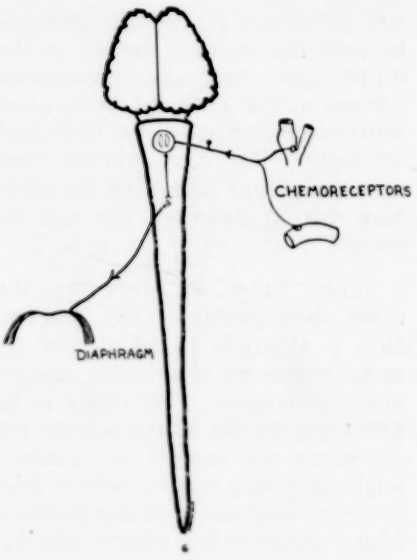


Fig. 5.—A respiratory center which is driven by impulses from the chemoreceptors; these are the most rugged and constitute the respiratory drive in adverse circumstances.

Fig. 6.—Paradoxical respiration; the center is driven by impulses from the chemoreceptors, but only the diaphragm responds to impulses from the center, as the intercostals are now paralyzed.

ation anesthesia, in high spinal anesthesia, and after the administration of curare.

Not only does the anesthetist have to contend with the depression due to premedication and anesthetic agents, but also, on occasion, the position desired by the surgeon may contribute further difficulty. The steep Trendelenburg, the gallbladder, lateral, and kidney positions, with rests, are known to interfere with respiratory exchange. The anesthetist who can maintain a satisfactory ventilation in the patient in spite of premedication, anesthesia, and difficult surgical position deserves a great deal of credit.

Circulation

I. Heart

The anesthetist determines the action of the heart by watching the patient's pulse. Although the heart beat is not dependent upon nervous impulses, it is regulated by them. The cardiac center, which is located in the medulla, can be affected by impulses from higher centers and from numerous receptors, just as readily as can the respiratory center. These incidental influences have not been indicated in figure 7, which depicts instead the source of the impulses which are especially important in the minute-to-minute regulation of the heart's activity. These impulses arise from pressorecep-

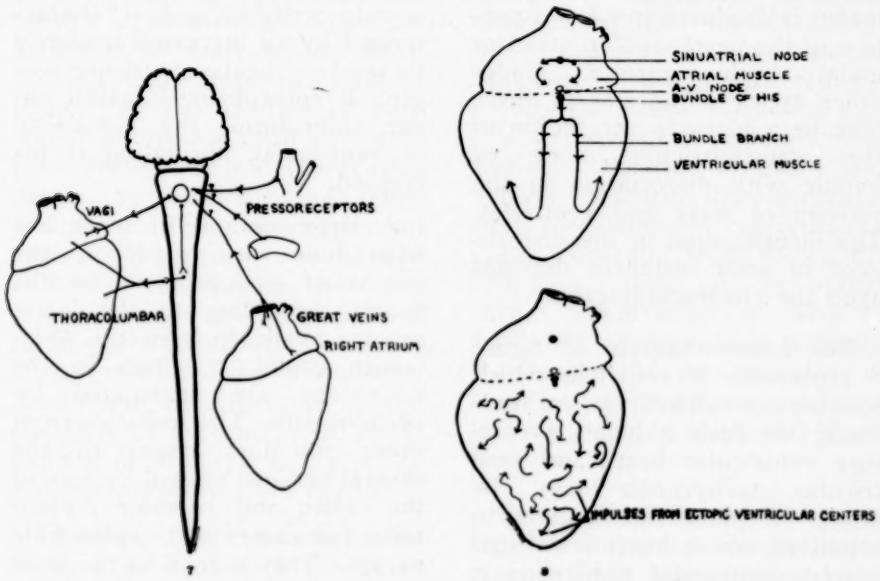


Fig. 7.—The cardiac center in the medulla is shown; on the right are the afferent pathways from the pressoreceptors, and on the left, the efferent pathways which carry impulses from the center to the heart.

Fig. 8.—In the upper diagram the path of the normal beat from the sinuatrial node to ventricular muscle is shown. In the lower diagram the pacemaker is no longer discharging; impulses are arising at random in the ventricle, and fibrillation results.

tors in the carotid and aortic sinuses, right atrium, and great veins. Changes in pressure in these sensitive areas affect the activity of the cardiac center in such a way that the rate of the heart is speeded up or slowed and its force increased or weakened as needed to maintain relatively constant conditions within the circulatory system.

The efferent pathways from the cardiac center are shown. The vagus nerves are distributed to all parts of the heart except the ventricles; they weaken, slow, or stop the heart and constitute the inhibitory nerves to the heart. The thoracolumbar nerves are distributed to all parts of the heart, including the ventricles; they increase both the rate and force of the heart beat and constitute the augmentor nerves to the heart.

The beat begins in the sinuatrial node, which is the pacemaker under normal conditions. As shown in the upper diagram in figure 8, the beat spreads from this node over the atrial muscle to the atrioventricular node, and then passes over the bundle of His and its branches to the ventricular muscle. Under certain

types of anesthesia the pacemaker is displaced to a lower portion of the heart, and an atrial or nodal rhythm results. Under other types of anesthesia there may be a block in the region of the atrioventricular node or bundle with dissociation in the activity of atria and ventricles. The identification of the disturbance in such instances depends upon the electrocardiogram.

The lower diagram in figure 8 represents a ventricle which has been sensitized by an anesthetic; in such a heart, premature ventricular beats and ventricular tachycardia may be noted. If epinephrine is given to a patient whose heart is so sensitized, ventricular fibrillation is likely to occur. In ventricular fibrillation, impulses arise from all parts of the sensitized ventricular muscle, and effective beats are impossible; under these conditions, blood cannot be pumped from the heart, and death results.

Various agents affect the heart in different ways. Morphine stimulates the vagus nerves and slows the heart rate. The slowing of the rate makes possible discharges from ectopic centers, since the sinuatrial node is depressed. Ether displaces the pacemaker downward and thus leads to an atrial or nodal rhythm, as shown by the electrocardiogram. The barbiturates depress the vagus nerves; this, of course, leads to a faster heart rate, since the augmentor nerves are now unopposed. Under spinal anesthesia the augmentor nerves are inactive, and the unopposed vagus nerves produce a slow rate. Chloroform makes the heart muscle irritable and is characterized by an increased tendency to produce beats of ectopic origin. If epinephrine is given under chloroform, the possibility of ventricular fibrillation is increased.

A large amount of work has been done with respect to the effects of cyclopropane on the heart. According to the latest reports available from the Wisconsin group, receptors in the mesentery are stimulated by cyclopropane. The impulses from these receptors travel to the central nervous system by way of the celiac and superior mesenteric plexuses and splanchnic nerves. They ascend to the level of the hypothalamus or thalamus; from the former, efferent impulses pass downward in the spinal cord to the thoracic region, and then out to the heart over the augmentor nerves. In this manner, the ventricular muscle is reflexly sensitized under cyclopropane. Atrioventricular blocks, premature ventricular beats, and ventricular tachycardia indicate a sensitized heart; if epinephrine is given to such a patient, ventricular fibrillation may be precipitated.

Various methods have been used to prevent the sensitization of the heart under cyclopropane. Some investigators advocate the use of procaine which depresses the heart muscle itself; other investigators report that procaine may send the heart into ventricular fibrillation under certain conditions. The barbiturates are thought to depress the hypothalamus selectively; the use of

these drugs as premedication agents is claimed by some anesthetists to decrease the sensitization of the heart to cyclopropane. The combination of high spinal anesthesia with cyclopropane should prevent the sensitization by blocking the reflex pathways. The combination of ether, cyclopropane, and oxygen is known to decrease the onset of ventricular irregularities; a satisfactory explanation has not been given, to my knowledge. Additional investigations on the question of prevention of sensitization of the heart under cyclopropane anesthesia are needed.

Certain procedures are particularly associated with the onset of cardiac irregularities. These include opening the peritoneum, closing the peritoneum, traction on the gallbladder or uterus, exploration of the abdominal cavity by palpation, removal of the appendix, and removal of the thyroid gland. The anesthetist must be on the lookout for changes in the condition of the patient when the surgeon is carrying out any of these procedures.

II. Blood Pressure

The importance of the blood pressure level under anesthesia has long been recognized, and frequent determinations are imperative. In addition to the cardiac center, the vasomotor center in the medulla is important in the regulation of the arterial blood pressure. This center, like the cardiac center, is controlled largely by impulses from the pressoreceptors in the carotid and aortic sinuses, as indicated in figure 9a. Pressure changes in these sinuses stimulate receptors and give rise to impulses, which in turn reflexly modify the size of the blood vessels as required to maintain relatively constant arterial and venous pressures. The pathway from the vasomotor center to the spinal cord and from the thoracolumbar region of the spinal cord to the arterioles is shown in figure 9a. Maintenance of vasoconstriction in the arterioles is a factor of primary importance in the constancy of arterial blood pressure.

The permeability of the capillaries is important in the maintenance of a relatively constant blood volume, which in turn affects blood pressure. When the permeability of the capillaries is normal, water and electrolytes pass freely out and in, and the plasma proteins are retained within the vascular system, as indicated in the lower diagram on the right in figure 9a. When hypoxia supervenes, the permeability of the capillaries increases, and protein is allowed to escape into the tissue spaces, as shown in the lower diagram on the left in figure 9a. With the loss of protein, more plasma passes into the tissue spaces, and the volume of blood in the vascular system is insufficient to maintain an effective circulation. The loss of plasma is an important factor in the onset of surgical shock.

The cardiac output is likewise a factor of primary importance in the maintenance of a relative-

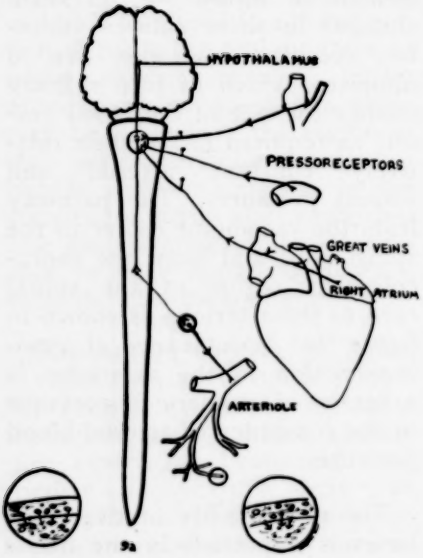


Fig. 9a.—The vasoconstrictor center in the medulla is shown. Impulses from the hypothalamus and from the pressoreceptors reach this center. It discharges to the spinal cord, and on by way of the thoracolumbar nerves to the arterioles especially in the skin and splanchnic areas. The diagram in the lower right section indicates the normal permeability of the capillaries; in the lower left section, increased permeability of the capillaries with leakage of protein into the tissue spaces is indicated.

ly constant arterial blood pressure. In order for the heart to pump out blood, it must receive blood from the various portions of the body. The mechanisms which play a part in the venous return are indicated in figure 9b. The striated muscles act as pumps; when they contract they force blood along, and the valves in the veins keep it from returning to the muscles when they relax. This muscular pump mechanism is exceedingly important in venous return.

The splanchnic nerves are important vasoconstrictor nerves; impulses which pass over these nerves from the spinal cord to the arterioles in the abdominal viscera prevent pooling of the blood in the abdominal and pelvic regions.

The spleen, especially in the dog, can change markedly in size; normally it acts as a storehouse for red corpuscles.

The liver is more important than is generally recognized as a blood reservoir. It can trap large amounts of blood or can force a considerable amount out of its sinusoids into the circulation by the differential action of sphincters at the entrance and exit of its blood vessels.

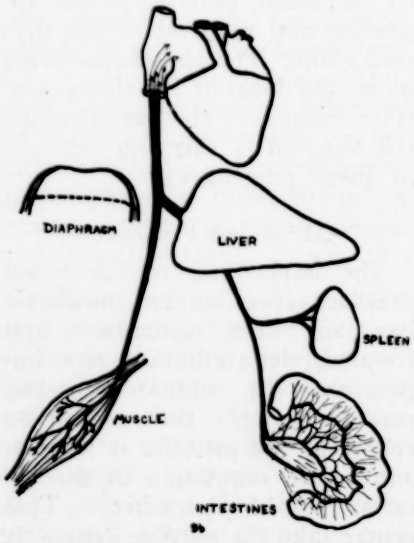


Fig. 9b.—Factors which are important in normal venous return.

The movements of respiration lead to changes in the negative pressure of the thoracic cavity; this negative pressure exerts a sucking or milking action on the large, thin walled veins in the thorax and prevents the stagnation of blood in the thoracic and abdominal veins.

Under anesthesia many of the important factors in venous return previously mentioned are altered, as indicated in figure 9c. The aim of the anesthetist is to give the surgeon the muscular relaxation he desires; this relaxation permits the accumulation and stagnation of blood in the venules and veins of the striated muscles.

There is dilatation of the blood vessels in the splanchnic and skin areas; this allows pooling and decreases the venous return to the heart.

Some anesthetics, and epinephrine, lead to a decrease in the size of the spleen; this forces more blood out into the circulation and at this time proves a benefit to the patient. Other anesthetics lead to dilatation of the spleen, which permits a large amount of blood to be trapped in this organ. The spleen may increase to three times its normal size.

The liver may allow large amounts of blood to accumulate, or by draining of its sinusoids, a smaller than normal amount may be present.

When respiratory movements decrease, as is usually the case under anesthesia, the venous return is slowed.

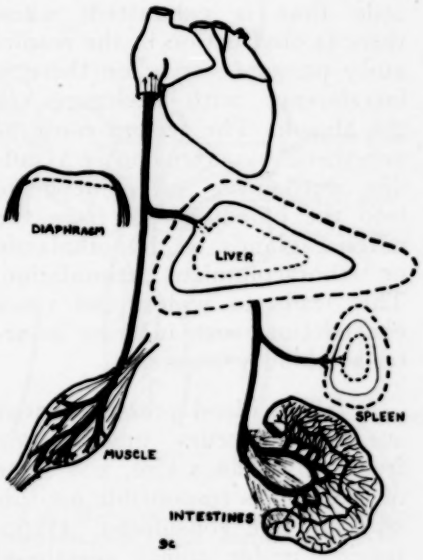


Fig. 9c.—Alterations in venous return under anesthesia. See text for explanation.

It is evident that many of the factors which are so important in prompt venous return normally may undergo marked alterations during anesthesia, and blood may be pooled in various regions instead of being returned to the heart; these changes lead to a decrease in cardiac output and a fall in blood pressure.

There are some occasions during anesthesia in which the blood pressure rises. This anesthetic hypertension is likely to be due to one of two causes. The first is an accumulation of carbon dioxide; an increase in tension of carbon dioxide in arterial blood stimulates the vasomotor center

and leads to widespread vasoconstriction with an increase in arterial blood pressure. Carbon dioxide accumulates when the soda lime is exhausted, when there is obstruction in the respiratory passages, or when there is interference with exchanges in the alveoli. The second cause of anesthetic hypertension is a sudden outpouring of epinephrine into the blood stream from the adrenal glands on hypothalamic or thoracolumbar stimulation. This leads to widespread vasoconstriction, with increase in arterial blood pressure.

A fall in blood pressure under anesthesia occurs much more frequently than a rise, and some of the factors responsible for this will now be considered. Hypotension under spinal anesthesia is the usual finding. It has been determined by investigation that it is the pooling of the blood on the venous side of the circulation which is largely responsible for hypotension under spinal anesthesia. Blood accumulates in the venules and veins of the striated muscles and in the spleen, which dilates to about three times its normal size. Both tend to decrease the venous return; the cardiac output falls, and the arterial blood pressure is bound to fall. On the arterial side of the circulation, two factors contribute further to this fall in arterial blood pressure; one factor is the lack of vasomotor impulses due to paralysis of the thoracolumbar roots by the anesthetic, and the other factor is the lack of epinephrine, since none is released when the thoracolumbar roots are inactive.

In addition to the aforementioned factors on the venous and arterial sides of the circulation, the loss of blood itself, or hemorrhage, leads to a fall in arterial blood pressure. If there is a decrease in blood volume, blood pressure cannot be maintained. This is especially serious during spinal anesthesia in which the compensatory mechanisms available to the normal individual are lacking. If hypoxia is present for any length of time, the capillaries become more permeable, and protein escapes into the tissue spaces. This upsets the mechanism for normal exchange of fluid between the vascular and interstitial spaces, and one

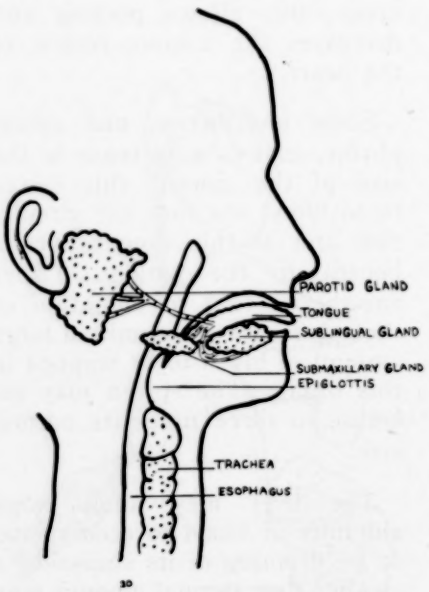


Fig. 10.—Normal secretion of saliva and mucus along the upper alimentary and respiratory tracts.

"bleeds to death" into his own tissue spaces, as it were. This fluid which is lost from the vascular system owing to increase in permeability of the capillaries is no more available to the individual than if it were lost by hemorrhage. This is the condition which prevails in surgical shock. It cannot be emphasized too strongly that a relatively moderate hemorrhage which can be readily tolerated by a normal individual may prove disastrous to a patient under anesthesia since his compensatory mechanisms are impaired. When one lacks the compensatory mechanisms, positional changes, like hemorrhage, may prove difficult for the patient under anesthesia.

GASTROINTESTINAL SYSTEM

Since this system is less important to the anesthetist than the foregoing ones, less attention will be devoted to it. The secretions of saliva and mucus by the glands along the alimentary and respiratory tracts are shown in figure 10. These secretions are markedly increased under anesthesia, as shown in figure 11, and a patient may literally drown in his own secretions if no premedication is given. The effect of giving atropine or a similar drug before anesthesia is induced is indicated in figure 12; the decrease in the amount of secretion is marked.

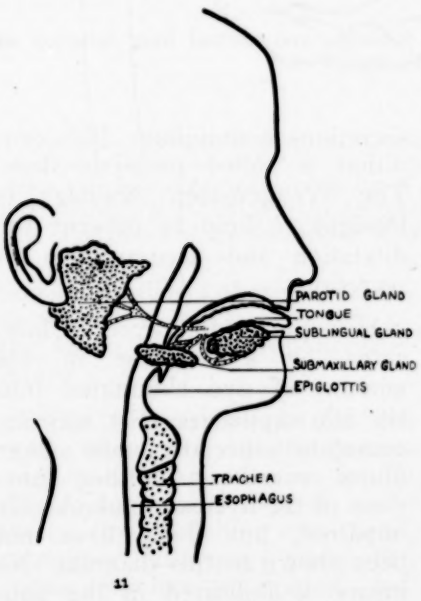


Fig. 11—Increase in secretion under anesthesia without premedication.

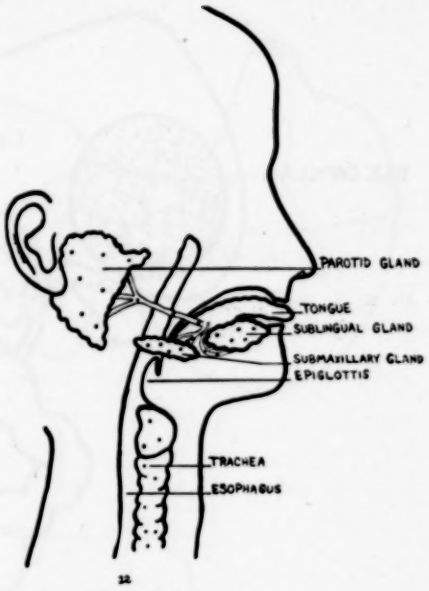


Fig. 12.—Decrease in secretion after premedication with atropine and related drugs.

In figure 13 the appearance of the stomach and intestine under normal conditions is shown. The smooth muscle is in tone, and the secretions are normal in amount. The liver cells are shown secret-

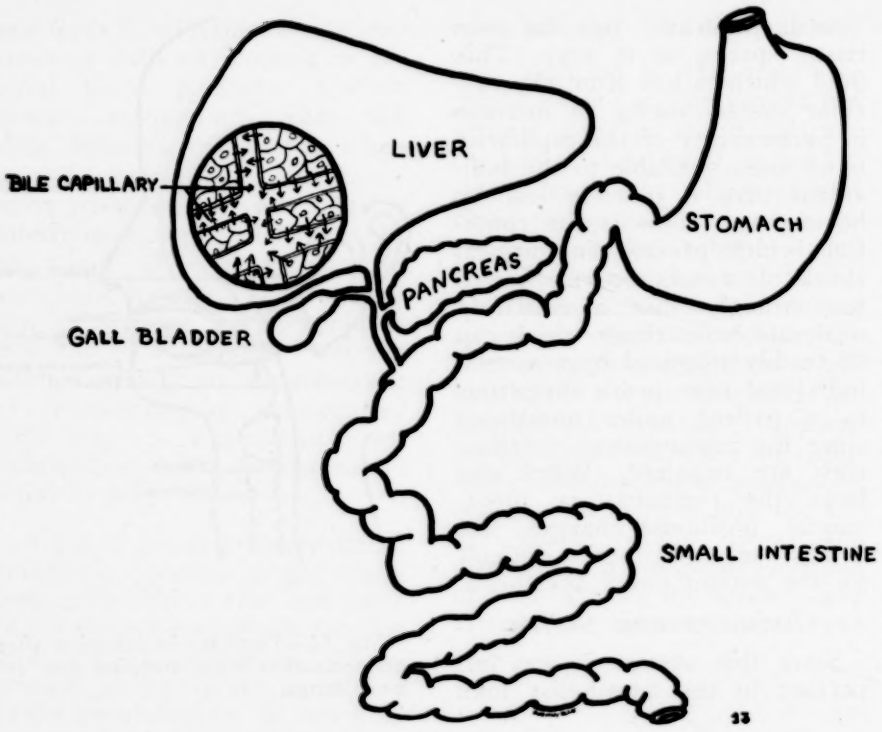


Fig. 13.—Normal tone in stomach and intestine and normal liver function as shown by the excretion of dye into the bile capillaries.

ing dye from the blood into the bile capillaries; this is the basis of one of the liver function tests.

Under anesthesia several changes may occur in the alimentary tract, as indicated in figure 14. The tone of the stomach musculature may be abolished; this permits dilatation. The greater the dilatation which occurs, the greater the amount of secretion formed. With dilatation and accumulation of secretions, the circulation and respiration suffer interference.

A commoner occurrence is loss of tone in a part of the intestine. This portion dilates and secretions accumulate. This condition is called paralytic ileus. The Wangensteen drainage is thought to help to prevent the dilatation and accumulation of secretions.

Damage to the liver is indicated by a decrease in the amount of dye eliminated into the bile capillaries. Fat may accumulate, especially under chloroform anesthesia. Other functions of the liver are undoubtedly impaired, but these have not been shown in this diagram. No injury is indicated in the pancreas; that it suffers some damage is shown by the fact that

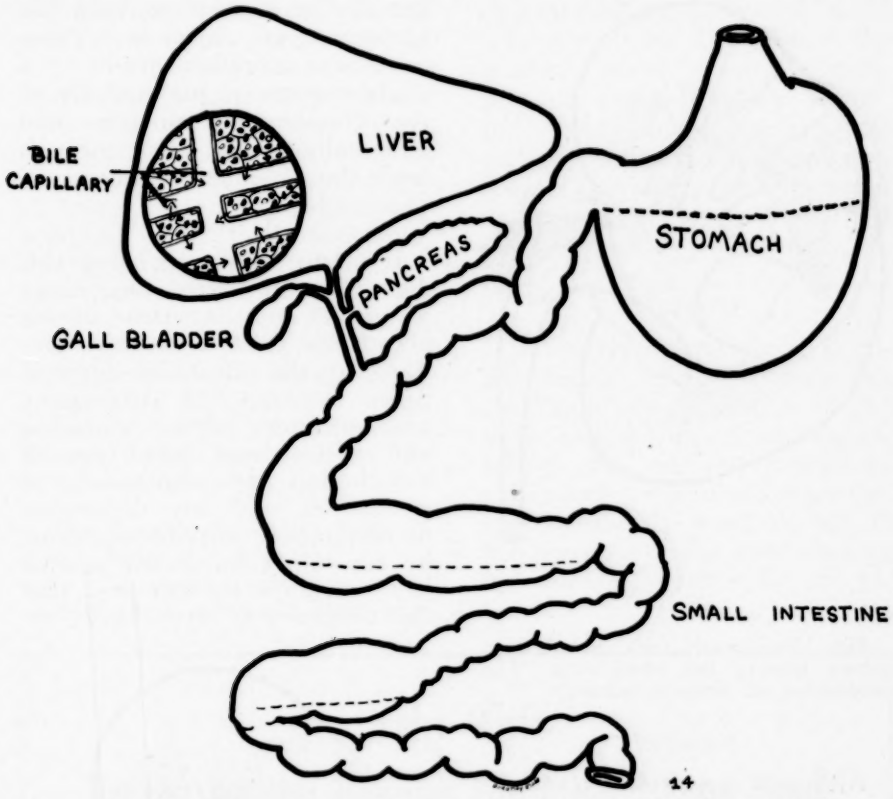


Fig. 14.—Changes during anesthesia. The stomach is dilated and excessive secretions have accumulated in it. A portion of the intestine is dilated and quiescent, with secretions accumulated in it. This represents paralytic ileus. The liver damage is indicated by a decrease in the amount of dye excreted into the bile capillaries and by the accumulation of fat droplets.

under anesthesia there is an increase in the amylase content of the blood, a sign of pancreatic injury.

Most general anesthetics decrease the activity of the gastrointestinal tract, so far as motility is concerned. Spinal anesthetics, by abolishing the thoracolumbar influence, leave the vagus impulses unopposed, and in general these increase the motility of the tract. Evacuation may occur after administration of spinal anesthesia. Vomiting is frequent under any type of anesthesia; it may be due to stimulation of the vomiting center in the medulla, accumulation of secretions in the stomach, irritation of the mucosa, hypoxia, and many other factors. The anesthetist must prevent the aspiration of vomitus and the excessive accumulation of secretions; other disturbances of this system do not fall in the realm of anesthesia.

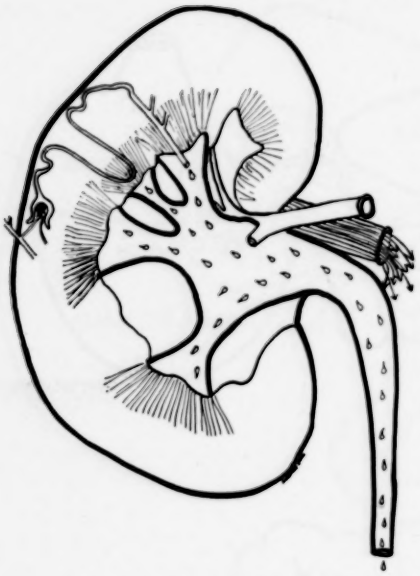


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Fig. 15.—Normal renal blood flow, shown leaving the renal vein. The production of urine is normal.

Kidneys

Although the alteration of kidney function during anesthesia is not of immediate concern to the anesthetist, the major changes will be called to the attention at this time. Figure 15 illustrates the normal blood flow and production of urine by the kidney. Under anesthesia, as indicated in figure 16, there is a decrease in renal blood flow and oliguria. The former is one of the factors responsible for the latter. Another factor is the increase in the amount of antidiuretic hormone secreted by the posterior lobe of the hypophysis. This increases the reabsorption of water by the kidney tubules, which in turn leads to oliguria. Albuminuria is often noted after anesthesia; it may be due to the decrease in blood flow, with the accompanying hypoxia; these conditions together lead to a slight increase in permeability of the glomerular capillaries and allow albumin to pass through from the plasma into the urine.

Hypoxia

It seems fitting to bring this discussion on altered physiology of normal body functions during anesthesia to a close by summing up the effects of hypoxia. Since hypoxia may accompany premedication with morphine and barbiturates, any type of anesthesia, any depression of circulation, and any depression of respiration, anesthetists must be constantly on guard against it. You know as well as I that the tongue may drop back; se-

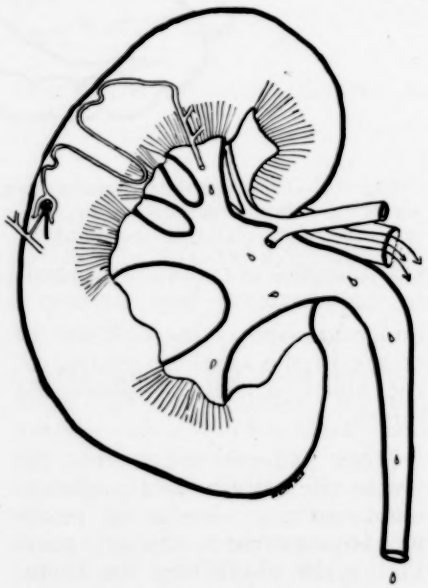

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Fig. 16.—Under anesthesia the renal blood flow is markedly decreased, and oliguria is evident.

cretions may accumulate in the respiratory passages; vomitus may obstruct the passages; the intercostal muscles may become paralyzed; the respiratory movements may be limited (owing to depression of the center, the use of rests, steep Trendelenburg position, or tired assistants' leaning on the chest); there may be a barrier to diffusion in the alveoli; there may be failure of the transport system (anemia); or a disturbance of tissue respiration (after sulfones). It seems impossible to list all of the conditions which may lead to hypoxia during anesthesia.

The anesthetist must keep in mind that the cerebral cortex may be irreparably damaged within a few minutes; there may be convulsions, increased intracranial pressure, dilatation of the cerebral blood vessels, depression of the respiratory center, and many other detrimental changes in the body during hypoxia. Therefore, the anesthetist must prevent hypoxia and its attendant disturbances.

SUMMARY

Whenever an anesthetic is administered, changes occur in practically every system of the body, and the means of compensating for adverse conditions are greatly curtailed; therefore the anesthetist must do all in her power to make conditions as favorable as possible for the patient.

My Gift

To the Agatha Hodgins Educational *Loan* Fund

I hereby donate $_____to the Agatha Hodgins Educational Fund, which is "designed to extend financial assistance to graduate nurse anesthetists in obtaining further education and training to become qualified instructors, or to instructors who desire and need additional training to become better qualified."

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(Checks should be made payable to: American Association of Nurse Anesthetists, for Educational Fund, and sent to: American Association of Nurse Anesthetists, 22 E. Division St., Chicago 10, Illinois.)

NOTE: Highlights of Educational Fund Resolution appear on page 334 of this JOURNAL.

GENERAL CONSIDERATIONS IN INHALATION ANESTHESIA

Charles Duncan Anderson, M.D.*
Chicago

Successful anesthesia requires skilful application of the anesthetic agent and continuous watchful observation of the patient during the operation. This much is taken for granted, and usually our patients have a satisfactory convalescence after the operation and anesthesia. But in some patients in whom a complicating pathologic condition increases the risk of anesthesia, dependence on such standards will not always assure the anticipated results. Other things become important. The effects of the anesthetic agents on the tissues and organs of the body must be appreciated, and the choice of the individual agent must be based, in part at least, on a consideration of these facts. To a lesser extent the method of anesthesia is important. It is true that with experience one gains a "feel" of the various agents and methods which leads one to make a better choice. However, it is wise for all of us to stop from time to time and consider the effects of the various anesthetic agents and methods on the patient, particularly the patient in whom one or more pathologic conditions either are directly associated with the operation to be done or exist concomitantly. It is particularly important to make these evaluations since we are called upon more often now than formerly to provide anesthesia for complicated and daring surgical procedures to preserve the lives of patients during the first decade and the later decades of life. Thus, if we are to do this, we must have an understanding of the pharmacologic and physiologic effects of the agents and methods we are using.

Premedication

The first important consideration, and this applies to all patients who receive anesthesia, is the premedication which the individual patient requires. Premedication cannot be intelligently given on a routine order basis. The patient should be seen, and some estimation made of the requirements of premedication in relation to all of the circumstances concerning the individual. The use of opiates is helpful in relieving psychic disturbances and pain. A disadvantage of opiates is that they may cause a severe decrease in respiratory exchange. When opiates are used, the dosage must be graded to meet the individual requirements, to provide the beneficial effects anticipated and desired,

Read before the Tri-State Assembly of Nurse Anesthetists, Chicago, May 5, 1948.
*Attending Anesthesiologist, Chairman of the Department of Anesthesiology, Presbyterian Hospital of the City of Chicago; Associate Professor of Anesthesiology (Rush), University of Illinois Medical School.

and to keep undesirable effects at a minimum. For the aged patient, the dosage of opiates must be reduced, if the drugs are used at all, as it would be for a patient in the first decade of life. The use of the belladonna group of drugs is decidedly advantageous in controlling secretions during the induction phase, but the question of sensitivity to these drugs, particularly in the infant and child, must be considered.

The main advantage of the barbiturates in the preanesthesia period is in allaying the patient's fears with regard to the proposed operation. The use of barbiturates is of more value to the excitable patient than to the stoical patient. Primarily, premedication is used for the comfort and peace of mind of the patient. Yet, in addition, premedicants are definite aids to the anesthetist in producing anesthesia. By necessity, inhalation anesthetics reach the central nervous system and produce anesthesia by way of the respiratory and circulatory systems. Accordingly, when premedication is used purposely as an aid to the anesthetist, it must be gaged to produce only the desired beneficial effect and to keep to a minimum deleterious effects on the respiratory and circulatory mechanisms which impede the induction of anesthesia.

CHOICE OF ANESTHETIC AGENT

The choice of anesthetic agent for each individual case must be governed by the particular pharmacologic effects of the various agents at our disposal and by the altered physiologic processes due to disease in the patient. Today operations are being performed on an increasingly larger percentage of patients who belong to the older age groups. Frequently, the operations proposed for these individuals are extensive and serious. The effect of the degenerative processes of aging and the frequent presence of associated disease, particularly of the cardiovascular system, considerably increase the risk. Examples of operations for this type of patient are prostatectomy for benign prostatic hypertrophy and operations on the stomach and bowel for cancer. These patients frequently have hypertensive vascular disease, which may or may not be associated with coronary disease. The choice of anesthetic agent then must be made in the light of these facts, and the agent must be one which will be the least deleterious to the patient.

If nitrous oxide or ethylene is the anesthetic chosen, it must be used cautiously to avoid any degree of hypoxia. It is particularly important that at least a minimum of 20 per cent oxygen and preferably 25-30 per cent oxygen be made available to the patient at all times.

With ether anesthesia these patients usually do well. Alterations in blood pressure are minimal; there is usually some increase in heart rate, due to the specific action of the agent. Patients with cardiac disease usually do not suffer any injurious effects provided abundant oxygen is available to prevent hypoxia. Coronary arteries are dilated, assuring better nutrition

to the heart. The main disadvantages of ether are the long recovery period and the tendency to postoperative nausea and vomiting due to the action of ether in depressing gastrointestinal functions.

The use of cyclopropane for patients of this type must be considered carefully. Definite cardiac irregularity and the well known increase in heart irritability produced by cyclopropane may make this agent an unhappy choice. During cyclopropane anesthesia there is usually an increase in blood pressure, which is usually maintained until the anesthetic is discontinued. At this time, or shortly thereafter, hypotension of greater or less degree may develop. In these patients, particularly those with cardiovascular disease, this resulting hypotension that may occur is detrimental. Hypertensive patients are accustomed to a high blood pressure, and they do not stand a pronounced lowering of the blood pressure for any length of time. These effects can frequently be overcome by combining ether with the cyclopropane; irregularities of the heart are decreased, and there is less tendency to hypotension after withdrawal of the anesthetic agent.

It must be remembered that for the aged patient the amount of anesthetic agent used is much less than for the younger adult. Frequently these patients are successfully anesthetized by very minimal amounts. In addition, they do not tolerate deep anesthesia, and the anesthetist should follow the operation closely with the aim of providing deep anesthesia only as required. However, the depth of anesthesia should not be so light that it interferes with the surgeon's ability to perform good surgery in the minimum amount of time. It is perhaps superfluous to mention the necessity of maintaining an efficient soda lime absorption system and, when open methods of administration are used, of preventing the piling up of carbon dioxide. In the hypertensive patient, particularly, neglect of this may considerably increase the hypertension and possibly cause a cerebral accident. The operative bleeding associated with ether anesthesia is usually the result of increased carbon dioxide or of oxygen want, or of a combination of these two factors.

Patients with thyroid disease and particularly those showing manifestations of hyperthyroidism frequently belong in the group just discussed. In this type of case it is common for the surgeon to infiltrate the tissues of the anterior region of the neck with a local anesthetic while general anesthesia is produced with nitrous oxide or ethylene. This procedure is valuable in that it allows the patient to wake up before the incision is closed, and the voice can be tested to make sure that the recurrent laryngeal nerves have not been involved in the operation. An equally satisfactory procedure for thyroidectomy is the use of ether as the anesthetic agent. This procedure is particularly valuable in thyrotoxic states associated with a pathologic condition of the cardiovascular system.

The choice of anesthetic agent for the diabetic patient presents a problem. If relaxation is not a factor, nitrous oxide or ethylene is satisfactory. If relaxation is needed, more potent agents, such as ether or cyclopropane, must be used. Ether produces an increase in blood sugar, whereas cyclopropane causes very little change. This would indicate that cyclopropane is the agent of choice for the diabetic patient. However, from a practical viewpoint, the diabetic patient tolerates ether anesthesia very well, provided particular care is taken to control the diabetes in the preoperative and postoperative period. In these cases it is beneficial to use either nitrous oxide or ethylene with the ether to decrease the amount of ether used.

Frequently the pregnant patient requires surgery. If complete uterine relaxation is required, ether is the anesthetic of choice. Cyclopropane causes increased tone of the uterine muscles. The fetus is anesthetized through the placenta from the maternal circulation by both ether and cyclopropane, but some workers believe that the fetal oxygen supply is decreased when cyclopropane is used and that a normal fetal oxygen supply is maintained when ether is used.

For surgery in the presence of pulmonary disease, it is a moot question whether one inhalation agent is superior to another. It is most important to prevent the accumulation of secretions in the pulmonary tree during the operation and in the postoperative period. It is well to have the patient clear his tracheobronchial tree by postural drainage and coughing before the anesthetic is given. During the operation secretions should be removed by suction. As a rule, some degree of the Trendelenburg position is helpful in causing bronchial and tracheal secretions to flow from the bronchial tree into the oropharynx, where they are readily accessible for aspiration by suction catheter. In the postoperative period an early return of reflexes and consciousness is beneficial, since with an early return of the cough reflex the patient can once again cough up secretions. The anesthetic gases have the advantage of an early return of reflexes and consciousness in the postoperative period, whereas when ether is used, the recovery period is prolonged. However, ether may be used in these cases, particularly if it is combined with the gases. An attempt should be made to conduct the anesthesia in such a fashion that minimal amounts of ether are used. Ether should be withdrawn as soon as feasible and anesthesia maintained by gases only in the terminal part of the operation.

The choice of anesthetic agent for splenectomy is an excellent example of how the patient is benefited by the anesthetist's knowledge of pharmacology. Ether is the anesthetic agent *par excellence* for this type of surgery. The effect of ether on the spleen is to contract the spleen, thus forcing out into the circulation the reservoir of blood which it contains. This is the equivalent of giving the patient a transfusion, only in this case it is a

transfusion of the patient's own blood. Some of the other anesthetic vapors possess this property to a degree, but in the anesthetic gases, including cyclopropane, it is entirely lacking.

General Considerations

I should like to draw attention to some other considerations of a more general nature. In inhalation anesthesia an adequate supply of oxygen must always be guaranteed to the patient, and the airway must be kept patent to allow the patient to benefit from the oxygen supplied. Any degree of hypoxia not only will prolong the convalescence of the patient but may very well incapacitate the patient or endanger his life through cerebral damage. It is well to remember that an increase in heart rate may be the only sign that the anesthetized patient requires more oxygen. Considerable hypoxia may be present without any clinical evidence of cyanosis. Even in the presence of an apparently adequate supply of oxygen, the respirations may be depressed just enough to create an oxygen deficit. A safe rule is to err on the side of providing too generous a supply of oxygen while maintaining a patent airway.

Another factor of importance pertains to emergencies in which a patient requires an operation soon after a meal. If general anesthesia is used, vomiting may occur, and the aspiration of vomitus into the respiratory tree is an ever present danger. Usually introduction of a stomach tube is of little value, since food particles block the lumen; however, unsuccessful attempts to insert the stomach tube are frequently helpful since they may initiate vomiting of the possibly dangerous stomach contents.

Finally, I would like to suggest the value of inserting a Levine tube through the nose into the stomach for most intraperitoneal operations. The tube is inserted before the operation, and all stomach secretions are aspirated. Aspiration is repeated at intervals during the operation, as well as during the postoperative period. The patient usually recovers from the anesthesia without nausea or vomiting. Also the reduction in abdominal distention during the postoperative period adds considerably to the patient's comfort.

Summary

An attempt has been made to point out some of the effects of various inhalation anesthetic agents and methods in the presence of pathologic states. The importance of adequate oxygenation of the tissues during anesthesia has been stressed, and mention has been made of tachycardia occurring during anesthesia as being a possible indication of inadequate oxygenation. Two suggestions for the control of nausea and vomiting during surgery and in the early postoperative period have been offered.

The anesthetic needs of the patient with consideration of his individual associated pathologic condition must not be minimized. The anesthesia should be as well adjusted to the physical status of the patient as a fine suit designed by an outstanding tailor.

CLINICAL ASPECTS OF ANESTHESIA FOR THE DIABETIC PATIENT

Daniel B. Marcus, M.D.*
Detroit

The alarming rate at which diabetes appears to be developing in the population of the United States makes it urgent that critical interest in the nature of this disorder be aroused. It is imperative that knowledge of the character of diabetes be disseminated among those who will inevitably be called upon to render their skilled services, since increasing numbers of individuals afflicted with this disease will require surgical treatment.

STATISTICS

This disease, which was twenty-seventh in the mortality tables at the turn of the century, in 1943 ranked eighth among the causes of death in the United States.[1] Other interesting statistics indicate that probably 1 in every 150 persons in this country has diabetes,[2] which implies that about one million individuals are so afflicted. On the basis of actuarial data, Spiegelman and Marks[3] estimated that 50,000 new cases of diabetes developed in 1940. According to Marks, a steady yearly increment brought the number of new cases in 1947 to 55,000. He predicted that by 1950, the number of diabetics would increase 18 per cent, while the whole population would advance but 9 per cent. The probabilities, statistically computed, applied to the 1946 population by Marks, indicate that 4,125,000 persons, or 2.9 per cent of this population, will eventually become diabetic, and that females will predominate in a ratio of almost 2 to 1 over the males.[4]

The increasing incidence of diabetes is authoritatively attributed to a number of factors, and these appear collectively or individually to serve in the predisposition to, if not in the actual production of, this disease. Chief among these are (1) heredity, (2) overnutrition, (3) obesity, (4) improvement in the diagnosis of the disease, and (5) lengthening of the life span of our people.

Evidence that the potentiality for diabetes to develop is inheritable as a mendelian recessive characteristic was abundantly set forth by White and Pincus.[5]

Read before the Tri-State Assembly of Nurse Anesthetists, Chicago, May 4, 1948.

*From the Diabetic Service, The Grace Hospital.

1. Joslin, E. P.; Root, H. F.; White, P.; Marble, A., and Bailey, C. C.: *The Treatment of Diabetes Mellitus,* ed. 8 (Philadelphia: Lea and Febiger, 1946) p. 27, table 3.

2. Joslin, E. P.: Diabetes mellitus. New England J. Med. **324**: 478, April 4, 1946.

3. Spiegelman, M., and Marks, H. H.: Age and sex variations in the prevalence and onset of diabetes mellitus. Am. J. Pub. Health **36**: 33, Jan. 1946.

4. Marks, H. H.: Recent statistics on diabetics and diabetes. Med. Clin. North America, March 1947, p. 2.

5. Joslin, E. P.; Root, H. F.; White, P.; Marble A., and Bailey, C. C.: *The Treatment of Diabetes Mellitus,* ed. 8 (Philadelphia: Lea and Febiger, 1946) chap. 3, p. 57.

Overnutrition and obesity are directly related to the increasing incidence by the comparison of food consumption in each nation with its mortality rate from diabetes. The people of the United States, the best fed in the world, have the highest death rate from diabetes.[6] Indeed, Joslin[7] drew attention to the apparent relation of the increasing prevalence of diabetes to the decrease in requirements for energy output. A major factor cited by him is the development and utilization of labor-saving devices, and consequent reduction in the number of hours in the working day with the resultant increase in time for leisure, while excessive eating habits are retained. Improvement in the diagnosis and treatment of the disease is understandably contributing to the greater frequency of its occurrence. This, in great measure, is also responsible for the increase in life expectancy and has caused a shift in the mortality rate, so that it is now greatest in and beyond the fifth decade of life. Dissemination of information to the public in all matters of health and voluntary periodic physical examinations are serving to bring the patient to the attention of the physician, and accordingly diagnoses are made earlier, as well as more frequently. Lengthening of the average life span is increasing the opportunity for diabetes to develop in more individuals. It has been pointed out that since 1900 the total population has increased 70 per cent, but that the increase in persons beyond the age of 45 years is 144 per cent and that the greatest frequency of onset of this disease occurs in the fourth and fifth decades of life.[8]

Pathogenesis and Pathologic Physiology

The metabolic and physiologic disturbances which characterize diabetes do not readily lend themselves to simplification for purposes of explanation, nor can it be stated with certainty that the correct or entire explanation is known at this time. Diabetes is considered to be a disease of metabolism, in the presence of which the body is incapable of metabolizing food in a normal manner. Protein and fat as well as carbohydrate cannot be properly utilized owing to a relative or absolute insufficiency of insulin. When untreated, or in the absence of adequate treatment, diabetes manifests itself through an inability to maintain the blood sugar level within normal limits, consequent to which, a greater or less degree of glycosuria may occur. Upon development of glycosuria, the characteristic train of symptoms—polyuria, thirst, hunger, weakness, loss of weight, pruritus, and alteration of visual acuity—becomes evident to a degree variable with patients. Control of the disease depends upon a satisfactory response by the patient to proper dietary restriction and, when necessary, to the administration of insulin in the needed amount at the re-

6. Joslin, E. P.; Dublin, L. I., and Marks, H. H.: Studies in diabetes mellitus: II. Its incidence and factors underlying its variations. Am. J. M. Sc. **187**:433-456, April 1934.

7. Joslin, E. P.; Root, H. F.; White, P.; Marble, A., and Bailey, C. C.: *The Treatment of Diabetes Mellitus*, ed. 8 (Philadelphia: Lea and Febiger, 1946) p. 68.

8. Idem. p. 92.

quired intervals. Under ideal conditions the patient exhibits control of the disease by maintenance of the blood sugar level within tolerable limits with a minimum of or no glycosuria; children are enabled to grow, and adults maintain ideal weight.

When hyperglycemia and glycosuria are unchecked because of lack of, or inadequate, treatment, all the described symptoms and signs of the disease are intensified. The constant and increasing glycosuria results in aggravation of dehydration by withdrawal of water from the body stores causing great thirst and polyuria. The continuing loss of carbohydrate in the form of sugar in the urine deprives the body of a source of substance essential for metabolic needs, causing depletion of glycogen stores. Insufficiency of available carbohydrate compels the body to use its own fat and to some extent its protein for metabolic purposes. Incomplete oxidation of fats yields an excessive amount of ketone bodies at a rate beyond which they can be utilized for energy production in the body. In the process of the development of acidosis, the disturbance of electrolyte balance in the body follows from the fact that, although some of the ketone bodies are excreted in the urine, as weak acids or after neutralization by ammonia, and some through respiratory exchange, a considerable amount displaces carbon dioxide from bicarbonate of the plasma, while a portion is neutralized by the buffering action of the blood proteins. The overproduction of ketone bodies becomes excessive to the point that fixed base is finally required, causing loss in the urine of sodium and chloride which the body itself is incapable of replacing. Potassium and phosphorus are known to be concomitantly reduced during the process of development of acidosis. The air hunger, first described by Kussmahl, is the direct result of a lowering of the carbon dioxide-combining power of the blood through these changes. The inability to utilize carbohydrate and the compensatory use of fats and proteins cause the loss of weight and the progressive weakness experienced by the untreated patient. If acidosis is permitted to continue, the ketone bodies, which have the properties of anesthetic agents, cause drowsiness, then stupor, and finally coma.

COMPLICATIONS

There can be no doubt that a significant relationship exists between diabetes and arteriosclerosis. The latter is unquestionably excessively prevalent in diabetics and, furthermore, not only tends to appear earlier in them, but advances at a more rapid rate with the production of more profound disturbances than in nondiabetics. Although coma is now responsible for only one twentieth of the deaths it formerly caused, the mortality from arteriosclerosis has tripled. The statistics presented by competent authorities are in substantial agreement that the prevalence of cardiovascular-renal complications accounts for the majority of the deaths of diabetics. An analysis of 651 deaths among

Joslin's[9] patients between 1944 and 1947 revealed that 67.4 per cent were due to cardiovascular-renal complications. In autopsies on 606 diabetics, Bell[10] found that a cardiovascular-renal pathologic condition caused death in 36.7 per cent and that 66.3 per cent died from some complication related to diabetes. Among Colwell's[11] patients acidosis was the most frequent complication in the first three decades, thereafter tapering off rapidly. In contrast, the incidence of vascular complications among them appeared as early as at the beginning of the second decade and increased persistently through successive age groups. Hypertension appeared earlier in these patients as well as more frequently than in control groups. In autopsy material examined by Bell,[12] the relationship between hypertension, arteriosclerosis, and diabetes was strikingly evident. The proportion of deaths from cardiac disease among Joslin's[9] patients between 1944 and 1947 was 44.5 per cent as compared to the rate of 6.1 per cent between 1898 and 1914. According to Boyd,[13] even in his young diabetic patients, retinopathy was likely to appear early, particularly among those in whom the disease had not been controlled satisfactorily. Dolger[14] insisted that good control of the diabetes in his young patients did not protect them from retinal damage, but that the frequency of occurrence appeared proportional to the duration of the disease.

9. Idem. p. 229, table 6.

10. Bell, E. T.: *Renal Diseases*, ed. 5 (Philadelphia: Lea and Febiger, 1946) p. 374, table 62.

11. Colwell, A. R.: *Diabetes Mellitus in General Practice* (Chicago: The Year Book Publishers, Inc., 1947) pp. 269, 275.

12. Bell, E. T.: *Renal Diseases*, ed. 5 (Philadelphia: Lea and Febiger, 1946) p. 375.

13. Boyd, J. D.; Jackson, R. L., and Allen, J. H.: Avoidance of degenerative lesions in diabetes mellitus. Proc. Am. Diabetes A., No. 1, 97-110, 1941.

14. Dolger, H.: Clinical evolution of vascular damage in diabetes mellitus. Bull. New York Acad. Med. **22**: 482, 1946.

Etiologic Factors

That the defect in diabetes is not simply a matter of too little insulin for the amount of food ingested is made readily apparent upon consideration of the results of investigation into the nature of the abnormal physiology involved. Long[15] propounded the theory of endocrine control of carbohydrate metabolism. His concept was substantiated in part by Houssay,[16] who demonstrated that diabetes produced in frogs and other animals by pancreatectomy can be prevented or ameliorated by removal of the hypophysis and adrenals. Cori and his co-workers[17] isolated and identified hexokinase as the enzyme essential in facilitating the interaction between adenose triphosphate and insulin in the carbohydrate metabolic cycle, as well as in proving the inhibiting effect of anterior pituitary and adrenal extracts upon the reaction. Methods for the experimental production of diabetes, besides pancreatectomy, were demonstrated (1) by Young[18] through the injection of anterior pituitary extract into animals,

15. Long, C. N. H.: Endocrine control of carbohydrate metabolism and its relation to diabetes in man. Proc. Am. Diabetes A., no. 2, 97-116, 1942.

16. Houssay, B. A.: Carbohydrate metbolism. New England J. Med. **214**: 971-982, May 14, 1936.

17. Price, W. H.; Colowick, S. P., and Cori, C. F.: Effect of anterior pituitary extract and of insulin on the hexokinase reaction. J. Biol. Chem. **160**: 633, 1945.

18. Young, F. G.: Permanent experimental diabetes produced by pituitary (anterior lobe) injections. Lancet **2**: 372-374, Aug. 14, 1937.

(2) by Jacobs and Colwell[19] by the continuous intravenous infusion of glucose in dogs, (3) by Lukens[20] who injected glucose solution into the peritoneal cavities of cats, and, finally, (4) by the intravenous injection of the chemical substance alloxan, which causes permanent degeneration of the islet tissue of the pancreas.[21] Facilitated through the use of radioactive tracer substances, the work of Stetten[22] on carbohydrate metabolism and of Medes[23] on the modus operandi of acidosis undoubtedly opened the way to improvement in the understanding of this disease. This means for exploring physiologic and pathologic mechanisms, hitherto not possible, should materially help in the revelation of the nature of diabetes and may be the foundation upon which will rest the ultimate discovery of the cause, and perhaps the cure, of this as well as other diseases.

TREATMENT

Improvement in the treatment of diabetes after the discovery and clinical application of insulin and advancement of dietotherapy was eventually reflected in the reduction of the mortality from coma. Joslin[24] reported that, between 1898 and 1914, 68.3 per cent of all his patients died from coma. The incidence decreased to 41.5 per cent for the period 1914 to 1922, owing to improvement in dietary treatment by the application of Allen's principle of undernutrition. After 1922 when insulin was made available, there was a rapid decline in deaths from coma. In the ten year period between 1936 and 1946, 4.4 per cent of Joslin's patients died in coma, and this was the cause of 1 per cent of all the deaths in that period. Improvement has been most notable in children with diabetes, specifically in the life expectancy of the 10 year old diabetic, which increased from 1.3 per cent years in 1914 to 39.8 per cent years in 1938.[25]

The difference that insulin has made is strikingly apparent on comparison of the surgical mortality rates in diabetics before and after the discovery of insulin. John[26] reviewed the literature for the purpose of making such a comparison and found that 22 authors, who reported a total of 2,023 cases, had a death rate of 24.2 per cent prior to the discovery of insulin; after its discovery, 40 authors, who reported a total of 14,231 cases, had a mortality rate of only 5 per cent. These figures may be taken as an indication of the degree to which insulin made possible the performance of surgery with a pronounced minimi-

19. Jacobs, H. R., and Colwell, A. R.: Lesions in the pancreas and the anterior hypophysis with fatal acidosis following prolonged intravenous administration of glucose (in dogs). Am. J. Physiol. **116**:194, 1936.

20. Lukens, F. D. W., and Dohan, F. C., cited by Ricketts, H. T.: Does hyperglycemia harm the diabetic patient? Med. Clin. North America, March 1947, p. 22.

21. Dunn, J. S.; Sheehan, H. L., and McLetchie, N. G. B.: Necrosis of the islets of Langerhans produced experimentally. Lancet **244**: 484-487, April 17, 1943.

22. Stetten, D., Jr.: Studies in carbohydrate metabolism; altered rate of certain metabolic processes. Proc. Am. Diabetes A., to be published.

23. Medes, G.: Mechanism of the production of ketone bodies. Proc. Am. Diabetes A., to be published.

24. Joslin, E. P.; Root, H. F.; White, P.; Marble, A., and Bailey, C. C.: *The Treatment of Diabetes Mellitus*, ed. 8 (Philadelphia: Lea and Febiger, 1946) chap. 13, p. 420; fig. 12, p. 421.

25. Marks, H. H.: Recent statistics on diabetics and diabetes. Med. Clin. North America, March 1947, p. 4.

26. John, H. J.: *Diabetes: A Concise Presentation* (St. Louis: C. V. Mosby Co., 1946) p. 141.

zation of risk. These statistics cannot take into account the numbers of patients for whom operations could be advocated without any considerable added risk, whereas they previously had to be denied the benefit of surgery.

Anesthesia

Generally, the choice of anesthetic agent and the technic of its administration are largely determined on the basis of an appraisal of the presenting physical status of the patient as well as the preoperative diagnosis. In the surgical patient with diabetes, added factors of prime importance necessitate careful consideration. In the presence of an already disordered metabolic state, it is imperative to reflect upon the degree to which adverse effects may be anticipated from preoperative medication, anesthesia, and the physiologic disturbances incident to surgical intervention, both during the performance of the operation and in the postoperative period. It is essential that cognizance be taken preoperatively of the patient's age and state of nutrition with special regard for the presence of acidosis, dehydration, and the degree of debility. Deleterious influences on the state of diabetic control arise from those factors which tend to elevate the blood sugar, deplete the glycogen store, interfere with resynthesis of glycogen in the liver, cause dehydration, interfere with oxygenation, disturb the electrolyte balance, or alter vascular dynamics. The extent to which these untoward effects influence the progress of the patient depends in great measure upon the physical state, in all its aspects, at the onset. In the presence of diabetes, complications usually cause an increase in severity which adds to the difficulty of maintaining control. The mere act of restricting to bed a patient whose disease is ordinarily controlled by diet may impose need for some insulin. The patient whose disease is usually well controlled with an ample diet and an unvarying dose of insulin may, upon being confined to bed, require a reduction in diet and an increase in insulin for maintenance of control. The summation of the restriction of activity, of the physiologic alterations arising from the presence of a pathologic process, of the surgery performed for its alleviation, and of the administration of an anesthetic agent results in an increase in severity of the diabetes, variable in degree and duration. In addition to infection, any abnormal state which elevates the temperature and metabolic rate may be expected to make the diabetes, at least temporarily, more severe. The difference in effect produced by the pathologic condition varies with age, the nutritional state, and the control of the diabetes. It may be anticipated that children and thin adults will generally be inclined to have severe diabetes, almost invariably requiring insulin, and will be least able to tolerate fasting or loss of fluid from the body. In adults, at or above normal weight, the disease is usually easily controllable by dietary restriction, and insulin is less frequently required. Although starvation or fasting may be

utilized with benefit in the treatment of diabetic patients for whom this regimen is suitable (e.g., the obese patient), it is nevertheless to be noted that the point to which it may safely be carried is variable. When the glycogen stores in the liver and tissues have been exhausted, the body turns to the use of its own fat and protein as a source of carbohydrate. In diabetes the effect of acidosis of starvation may not be serious as long as the total metabolism is not increased as, however, it frequently is in the presence of hyperthyroidism, pregnancy, and infection, or as it is in children, and when there has been prolonged diarrhea or vomiting. In diarrhea and vomiting, the additional factors of fluid loss and interference with carbohydrate absorption serve to intensify greatly the process of acidosis.

The adverse effects upon the diabetic state induced by the performance of an operation and the administration of anesthetic agents are the combination of several elements:

1. The pathologic process itself increases the rate of metabolism (e.g., infection), thereby intensifying the severity of the diabetes as the effectiveness of insulin and diet in controlling the disease is concomitantly reduced. An increase in the glycemic level follows with attendant glycosuria, dehydration, and possibly a loss of electrolyte and fixed base, all of which tend to promote acidosis.

2. The administration of preoperative medication, the barbiturates and derivatives of opium, counted upon to facilitate anesthesia and the performance of the surgical procedure, is known to be capable of unfavorably affecting the diabetic control. In general, these drugs may depress liver function, hasten depletion of the hepatic glycogen store, prevent or delay resynthesis of glycogen, cause hyperglycemia, reduce the carbon dioxide-combining power, increase the rate of ketone body production, and tend to cause anoxia. Their action may be prolonged in the presence of liver damage. These effects are less pronounced or minimal when hypnotic or reduced doses of the drugs are used postoperatively.

3. The anesthetic agents themselves and the mode of their administration in combination with the use of preoperative medication may tend to favor the production of anoxia, decrease the carbon dioxide-combining power, cause hyperglycemia, rapidly deplete the liver of its glycogen store, prevent resynthesis of glycogen, be hepatoxic (as in the case of chloroform and divinyl ether), and frequently cause postoperative nausea and vomiting. Specific untoward alterations affecting metabolism, the liver, and blood are considered to be minimal with the use of the least toxic of the local anesthetic agents.

4. Finally, the direct and indirect effects of the performance of the operation itself must be considered. The extent of physiologic disturbance created depends upon the tissues and organs involved, the time required for the operative procedure, the duration of anesthesia, and the

elements of shock and anoxia.

The actual harm to the diabetic patient from preoperative medication and anesthesia is dependent upon the injudiciousness with which they are employed. In the administration of preoperative medication, the patient's well-being is safeguarded by the administration of minimal effective doses which will nevertheless enable the surgeon to perform to the best advantage, yet will affect the control of the diabetes as little as possible. It is of great importance to make use of drugs which are both detoxified and eliminated easily, and whose effects will not be prolonged unduely into the postoperative period, or tend to cause nausea and vomiting and prevent early feeding. In aged and debilitated patients, reduction in circulating blood volume, diminution in oxygenation of the blood, depression of respiration, and disturbances of metabolism which may result from anesthesia and surgery are not well tolerated in the presence of a circulatory system which has lost its ability to compensate owing to the presence of degenerative vascular changes. In the very young, and particularly in the debilitated patient or the patient with acidosis and the aged, with consideration of the prematurity and intensity with which arteriosclerotic complications may be present, it appears indicated to administer barbiturates and opiates in reduced doses preoperatively, or to reserve their use for clearly evident need during the course of the operation.

The consensus[27-35] bears out the common clinical impression that almost any form of anesthesia may be successfully adapted for use for the diabetic patient requiring an operation provided the facilities for adequate treatment of the diabetes are available. Generally, however, there is exception to the use of the more potentially toxic agents, including chloroform, avertin, ethyl chloride, ether, and d i v i n y l ether. Objection to these agents is based upon their recognized tendency to be damaging to the liver, promote most actively those processes which cause aggravation of the diabetes, produce acidosis, and be most likely to cause nausea and vomiting postoperatively. From the standpoint of the liability for adversely affecting diabetes, the employment of the less toxic local anesthetic agents with proper safeguards is preferable to the use of inhalation anesthetic agents. Of those administered by inhalation, the common gaseous agents have proved far safer than the volatile

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27. Joslin, E. P.; Root, H. F.; White, P.; Marble, A., and Bailey, C. C.: *The Treatment of Diabetes Mellitus*, ed. 8 (Philadelphia: Lea and Febiger, 1946) chap. 26, p. 680.

28. Colwell, A. R.: *Diabetes Mellitus in General Practice* (Chicago: The Year Book Publishers, Inc., 1947) p. 224.

29. Wilder, R. M.: *Clinical Diabetes Mellitus and Hyperinsulinism* (Philadelphia: W. B. Saunders Co., 1940) p. 214.

30. Sprague, R. G: Diabetes mellitus. *The Practice of Medicine* (Hagerstown, Md.: W. F. Prior Co., Inc.) **9**:120.

31. Cullen, S. C.: *Anesthesia in General Practice* (Chicago: The Year Book Publishers, Inc., 1946) p. 69.

32. Idem. p. 149.

33. Adriani, J.: *Techniques and Procedures of Anesthesia* (Springfield, Ill.; Charles C Thomas, Publisher, 1947) pp. 7, 59, 76, 81, 102, 144.

34. Mousel, L. H., and Weiss, W. A.: Comments on anesthesia. S. Clin. North America, Oct. 1945, p. 1078.

35. McKim, L. H., and Fowler, A. F.: Surgery and diabetes mellitus. S. Clin. North America, Oct. 1945, p. 1031.

anesthetic agents. The mode of administration of the inhalation anesthetic agents should be consistent with the best interests of the patient as well as with the needs of the surgeon. Limitations placed upon the surgeon by the anesthesia are obviously undesirable and are usually avoidable by careful choice of the agent or combination of agents and of procedures to be followed with consideration for eventualities.

The preference for gaseous anesthetic agents seems to depend upon the experience of the surgeon as well as the needs of the patient. The anoxic effects of ethylene, nitrous oxide, and cyclopropane incident to the production of anesthesia by them are a relatively serious contraindication to their use, especially for long surgical procedures, and for debilitated patients or patients with arteriosclerosis who have increased susceptibility to cerebral damage. Inasmuch as full, or larger than usual, doses of drugs for preoperative medication and the supplemental use of other agents are necessary to facilitate anesthesia with ethylene, nitrous oxide, and cyclopropane, their use may not appear desirable for long operations. Provision for an adequate oxygen supply during the administration of any of the inhalation anesthetic agents is essential. Cyclopropane is not recommended for use in the presence of disturbances of cardiac rhythm, and it is known to cause ventricular fibrillation, but it, nevertheless, is widely popular as an anesthetic agent. There is an almost unequivocal belief in its safety, to such an extent, in fact, that its administration, even without regard for contraindications to its use, is known to be common. It has been condemned by Mousel and Weiss[36] as "one of the most dangerous anesthetic agents in use today" because of its tendency to cause sudden changes which are difficult to combat and which may result in death unexpectedly. These serious situations may arise in the induction phase or occur after the anesthetic has been discontinued. The frequent development of postoperative psychosis after the administration of cyclopropane is cited by these authors. It is well known that cyclopropane is capable of adversely affecting vascular dynamics by provoking sudden changes in blood pressure and pulse rate, as well as of causing cardiac arrhythmias by increasing cardic irritability. In view of these known toxic effects and the potentiality for producing anoxia, it might be well to re-examine the question of the purported safety of cyclopropane as an agent of anesthesia. Its use for diabetic patients should be carefully considered in the light of the frequency with which they present arteriosclerotic complications and of the desirability of avoiding anoxia in them.

The inhalation agents for anesthesia of short duration, ethyl chloride and divinyl ether, as well as other volatile agents, chloroform and ether, should not be used in the presence of diabetes, because of their unfav-

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36. Mousel, L. H., and Weiss, W. A.: Comments on anesthesia. S. Clin. North America, Oct. 1945, p. 1078.

orable effects upon the disease and the fact that satisfactory and less noxious agents are available.

The intravenous anesthetic agents, with proper regard for contraindications to their use, are valuable for operations of very short duration or for supplementing or intensifying anesthesia produced by other means. However, particularly for patients at the extremes of age and for those with debility or acidosis, these agents, pentothal sodium and procaine, should be avoided. Furthermore, their should be prohibited in the presence of heart disease, hypotension, liver disease, kidney disease (which would prevent detoxication and elimination), and intracranial pathologic conditions. Their use should especially not be considered safe under conditions in which the anesthetist may not have complete control of the airway and constantly be able to observe the patient properly for premonitory changes.

Local anesthetic agents are adaptable for use under the many variable conditions which may be presented by the diabetic patient undergoing an operation. The modes of administration, infiltration, regional block, and spinal, afford means for providing anesthesia in the presence of the most serious physical states without any considerable added hazard if precautions and contraindications are faithfully observed. The use of this means of providing anesthesia should not be undertaken except by those having a thorough understanding of all its aspects. Procaine and metycaine are considered to be the least toxic of the drugs available.[31,34,37,38] The use of reduced dosages compatible with the duration of anesthesia desired is expedient for the diabetic patient, particularly in the presence of complications other than the condition requiring operation. Overdosage with drugs for preoperative medication is to be carefully avoided.

The four groups of substances which are useful in general surgery are likewise of value during the surgical treatment of the diabetic patient. They include (1) the nonanesthetic drugs, atropine and scopolamine, (2) the stimulating drugs, ephedrine, epinephrine, neosynephrin, metrazol, alphalobeline, etc., (3) curare, and (4) oxygen and carbon dioxide. The requirement for any of these supersedes any contraindication to their use for the diabetic patient undergoing an operation. The antagonistic effects they may have upon the disease are unimportant in relation to their possible benefit. The adverse effects which may be anticipated through their use can be minimized or avoided by the use of reduced doses compatible with the status of the patient, particularly of those patients in the extremes of age. Furthermore, these effects are easily nullified in the course of the application of the adequate treatment the patient will presumably be receiving.

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31. Cullen, loc. cit.

34. Mousel, loc. cit.

37. Lundy, J. S.: *Clinical Anesthesia* (Philadelphia: W. B. Saunders Co., 1942) p. 35.

38. Adriani, J.: *The Pharmacology of Anesthetic Drugs*, ed. 2 (Springfield, Ill.: Charles C Thomas, Publisher, 1947) p. 49.

The question of hypoglycemia, or an "insulin reaction" as it is commonly called, is vitally important, even though its occurrence during anesthesia is unusual. Most patients who come to surgery are generally possessed of a higher than normal blood sugar by virtue of any one, or a combination, of several factors. Difficulty in maintaining good control due to the pathologic process present (e.g., infection) and the administration of glucose preoperatively with insufficient insulin are the common causes. The administration of preoperative medication and the anesthetic agent and the performance of the operation tend to elevate the blood sugar level further. However, patients with abnormally low blood sugar levels due to overadministration of insulin, for one reason or another, may reach the operating room. The blood sugar may fall during the operation owing to the unanticipated effect, for example, of protamine zinc insulin given the previous day, plus an injection of quick acting insulin prior to the operation, which under the circumstances will constitute an excessive amount. Recognition of this state prior to the operation is facilitated by a blood sugar determination made within a half hour of the time of starting the operation. Otherwise, the clinical appearance of the patient must be relied upon as an indication of the presence of insulin shock. The skin will be cool and clammy, respiration will be quiet and somewhat slowed, the blood pressure will be lowered, and the patient will perhaps present a dazed appearance out of proportion to expectations. During anesthesia, recognition of this condition will understandably be much more difficult. It should be suspected, especially with the unaccountable appearance of profuse perspiration and coolness of the skin, a decrease in blood pressure, respiration becoming slower and shallow, twitching of facial muscles, or even convulsive movements. The immediate injection of glucose is indicated for the correction of this condition.

MORTALITY AND MORBIDITY

The mortality and morbidity rates for surgery of diabetic patients are considerably less today than in any previous period used for comparison. Further reduction is possible and can be accomplished by taking advantage of even the most trivial of the procedures which may be beneficially applied to the diabetic patient. In regard to the hazard faced by the diabetic patient who is confronted with the prospect of surgery, the degree of risk is minimized when adequate facilities are available for the proper treatment of the diabetes. The patient whose disease is well controlled before the onset of the illness requiring surgical intervention faces less risk of mortality or morbidity than the patient whose disease is uncontrolled or was not diagnosed preoperatively. The greatest hazard is faced by the patient whose diabetes is discovered during the operation or in the postoperative period. The best results are to be obtained under circumstances which will permit adequate preoperative preparation, even if the urgency of the

operation will allow only the beginning of treatment. Few surgical conditions require such immediate intervention that some delay, during which treatment can at least be started, is not permissible. With the knowledge of the situation under these conditions, vigorous treatment may be continued during the operation and into the postoperative period to the patient's advantage. Close adherence to these principles can materially assist in the reduction of mortality and morbidity in these patients. Although it may not be within the province of the nurse anesthetist to judge risk, nevertheless the poor physical state of the patient, the obvious lack of proper preoperative preparation, or tardy recognition of the diabetes imposes on her a moral obligation to draw attention to these defects before an operation is begun.

Summary

Because of the factors of a steadily increasing incidence of diabetes and the longevity of diabetic patients, greater numbers of patients with this disease will inevitably require surgical treatment. There is no longer the fear or hesitation which formerly stayed the hand of the surgeon, for since the advent of insulin and improved knowledge of treatment, as well as earlier recognition of the diabetes, the risk has been greatly diminished. Moreover, improvement in surgical technics, preoperative preparation, and postoperative care, and the application of sound principles in anesthesia contribute in equal measure to the success of surgical intervention for these patients. It is evident that, with the prospect of more diabetic patients' coming to surgery, a more widespread appreciation of the problems and an understanding of proper and adequate treatment are essential.

PREANESTHETIC MEDICATION FOR THE PEDIATRIC PATIENT

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During the past decade the rapidly expanding field of pediatric surgery has brought more younger patients to surgery for longer and more difficult operations. Elective tonsillectomy and adenoidectomy, emergency appendectomy, and traumatic surgery in children and infants no longer dominate the surgical picture. Correction of some congenital deformities requires surgical intervention in the first few hours or days of life, while correction of others may be deferred a year or more until the patient is better able to withstand the necessary surgical procedures. Cardiac surgery in young children, even in infants, is no longer a surgical curiosity. This progress is very gratifying, but it has brought with it many problems for the anesthetist. The choice of anesthetic agent, the technic of administration, the supportive therapy during anesthesia, the maintenance of adequate pulmonary function, and the use of preanesthetic medication are a few of the outstanding problems which the anesthetist must face in order that surgery may be successful.

Preanesthetic medication for infants and children has been a controversial subject for some time. On the negative side of the issue, many anesthetists and pediatricians believe that the drugs used are unnecessary and may even be dangerous to the patient. They quote with authority the statement appearing frequently in textbooks, that infants are excessively sensitive to sedatives, especially opiates, and that toxic, even fatal reactions, to the belladonna drugs are not infrequent. Some of those who object to sedation in children do so because they believe it is unnecessary in the preoperative period since the child is incapable of understanding, and that he soon forgets all about his unpleasant experience.

Recent studies provide us with answers to these chief objections to use of preanesthetic medication, namely, excessive depression from opiates, toxic reactions to the belladonna drugs, and the absence of any need for such medication. Mitchell and Nelson's *Textbook of Pediatrics*[1] suggests that, when there is an indication for morphine sedation, a 1 month old child weighing ten pounds should be given 1 mg.

Read before the Sixteenth Annual Meeting of the Pennsylvania Association of Nurse Anesthetists, Philadelphia, April 28, 1948.

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1. Nelson, Waldo E.: *Mitchell-Nelson Textbook of Pediatrics*, ed. 4 (Philadelphia: W. B. Saunders Co., 1945).

(gr. 1/60) of the drug. On this basis the infant receives 0.1 mg. morphine sulfate per pound body weight. Calculated for a 150 pound adult, a comparable dose would be 15 mg. (gr. 1/4) of morphine sulfate for sedation. From this, one can conclude that the infant is just about as tolerant of morphine as is the adult, based upon the quantity per pound. The general acceptance of oversensitivity of infants to morphine and its derivatives has probably arisen from the use of too large a dose, or possibly from the inaccurate administration of minute doses prepared from the standard hypodermic tablets.

Toxic reactions to the belladonna drugs, atropine and scopolamine, do occur, but they are infrequent and undoubtedly represent a true drug idiosyncracy. The so-called atropine or scopolamine flush, generally a scarlatiniform rash, is not evidence of a toxic reaction, but will accompany all adequate doses for preanesthetic medication. The toxic signs are excessive d r y n e s s of skin and mucous membranes, high fever, evidence of central nervous system stimulation, such as tremors, inco-ordination, even convulsions, and hallucinations. In over 3,500 administrations of atropine or scopolamine to infants and children, only three cases of toxic reactions were noted. One was evidenced by a temperature of 103 F. rectally in an infant who was receiving a transfusion at the time the drug was given; the other two were evidenced by hallucinations in children of 4 and 5 1/2 years, both of whom had also received barbiturates. None of these reactions required treatment.

In answer to the objection that children do not need preanesthetic sedation, an article by Levy, "Psychic Trauma of Operations in Children,"[2] should be read. There may, perhaps, be overemphasis upon the role of anesthesia in producing behavior problems in children, but until proved otherwise, the anesthetist must accept this responsibility. It is well to remember that the child who finds security in his parents and in his home may be separated from this security for the first time when he comes to the hospital. Not only the loss of security, but being cared for by total strangers and being forced against his will to face a strange and unpleasant experience are hardly reassuring. The child is little concerned with the nature of the operation to be performed, or what surgeon does the operation, but he is deeply concerned by the rather terrifying prospect of being rendered unconscious by noxious agents.

On the positive side of the issue there are many anesthetists who prefer to have their pediatric patients given preanesthetic medication in a fashion similar to adults. The same advantages that accrue to adults from suitable drugs in adequate dosage will be apparent in the pediatric patient. The lessening of apprehension, reduction of reflex excitability, and provision of adequate comfort and relief from pain in the immediate postoperative period are as important to infants and children as to adults.

2. Levy, David M.: Psychic trauma of operations in children. Am. J. Dis. Child. 69: 7, Jan. 1945.

Even more important than the aforementioned advantages is the reduction of an excessively rapid and exhausting respiratory rate. This is most often seen in infants who have not received premedication and who are given open drop ether without provision for the adequate elimination of carbon dioxide. Under such circumstances, the respiratory rate may approach or even exceed 100 per minute. This will quickly and needlessly exhaust the limited reserve energy of the infant, as well as exhaust the respiratory center. Exhaustion of the respiratory center which may result is probably an indication of far more widespread damage in the central nervous system. Undoubtedly, some of the advantages of anesthetic agents which do not cause hyperpnea are due to the diminished respiratory effort and conservation of the energy of the infant.

Opiates used for preanesthetic medication in the dosages suggested do not produce the soporific effect usually seen in adults. The children, as a rule, are awake but comfortable and show little evidence of fear when they arrive in the operating room. A further advantage of opiates in the preoperative period is reduction in the amount of anesthetic agent required, with a consequent widening of the margin of safety for the patient. The use of smaller quantities of the anesthetic agent permits a more rapid recovery of protective reflexes and consciousness and thereby reduces the amount of nursing care required in the immediate postoperative period.

Preoperative medication with opiates has one disadvantage: postoperative nausea and vomiting may occur. With large doses of morphine this is especially true. Demerol or other morphine derivatives such as dilaudid have been used, but these substitutes will not guarantee freedom from nausea and vomiting, and their action is less predictable than that of morphine. Persistent vomiting for twenty-four hours or more in this age group is a serious hazard, for it will result in rapid nutritional failure and chemical imbalance. For this reason, the dose of morphine should be kept at the minimum which is effective.

In large measure the nausea and vomiting usually seen with the administration of morphine is prevented by the use of barbiturates in the preanesthetic medication. The barbiturates are not analgesic in ordinary doses but produce good amnesia. They are excellent for allaying apprehension and fear. Moreover, the barbiturates are most valuable in preventing convulsive seizures which may occur during open drop anesthesia with a vinethene-ether sequence or gas-oxygen-ether by closed technics.

One of the belladonna drugs, either scopolamine or atropine, is essential for satisfactory preanesthetic medication. The tremendous advantage of having the respiratory tract free from secretions cannot be overestimated. Excessive secretions result in partial respiratory tract obstruction, and increased respiratory effort is necessary to overcome the obstruction. Furthermore, excessive frothy secre-

tions will be partially aspirated into the tracheobronchial tree and may be a major factor in the production of postoperative atelectasis and pneumonia.

Scopolamine is probably preferable to atropine for two reasons. First, the drying effect of scopolamine is greater than that of atropine in the same dose. This means that the desired result is achieved with smaller amounts of the drug when scopolamine is used. Secondly, the duration of action of scopolamine is twice that of atropine even when the latter drug is used in large doses. Another minor advantage of scopolamine over atropine lies in its action on the central nervous system. Possessing amnesic properties of its own, scopolamine depresses cortical activity, thereby enhancing the sedative action of morphine and the barbiturates without depressing vital functions. Atropine, however, does not possess amnesic properties to any degree, and its action on the cerebral cortex is purely stimulant.

In an occasional patient there may be a rise of a degree in rectal temperature after the administration of atropine or scopolamine. Such an elevation is of no consequence in view of the tremendous advantage of an unobstructed airway, free from secretions.

For infants and children as for adults, no hard and fast rules can be set for doses of preanesthetic medication. The drugs used and the dosage must be varied to suit the individual case. There are too many modifying factors to list them all, but among the more important are physical status, anesthetic agent selected, and surgical procedure to be performed.

The acutely ill newborn may need no sedation, whereas scopolamine is needed in order to lessen the burden of increased respiratory effort. The very husky 4 year old may tolerate a large dose of morphine which in the average 4 year old would produce profound depression. Cretins and Mongolian idiots tolerate morphine poorly and should receive very small doses or none at all. Except when sensitivity to the belladonna drugs is known to exist, all patients should receive either scopolamine or atropine. It is said that idiosyncrasy to these drugs is more likely to occur in infants with congenital cataracts. In such patients a small test dose should be tried twenty-four hours before preanesthetic medication is given. True idiosyncrasy to scopolamine or atropine will be apparent after a test dose of gr. 1/1,000, and the symptoms of hyperthermia and restlessness will be more readily controlled than after the usual preanesthetic dose.

The table of preanesthetic medication is the one most frequently used in the Children's Hospital of Philadelphia. Obviously, adjustment of dosage for the individual patient is often made. In the practical application of the data in the table, the weight of the patient is a far better guide to the amount of medication to be employed than is the chronological age. In general, a heavy, robust 4 year old,

weighing 50 pounds, will tolerate and, in fact, needs more sedation than a 5 year old whose weight is only 30 pounds. The dosage scale given here is the minimum which will provide adequate sedation for various age groups of infants and children. It will more often require adjustment by increasing the dose rather than decreasing it.

that of dissolving the 1/8 gr. tablet in 1 cc. sterile water in a tuberculin syringe and administering 1 minim. One cannot give an accurate dose by this means. Reasonable accuracy, however, can be achieved by the use of large volumes. In this case the 1/480 gr. represents 1/60 of the 1/8 gr. tablet. It is recommended that the 1/8 gr. tablet be dis-

SUGGESTED PREANESTHETIC MEDICATION FOR INFANTS AND CHILDREN

AGE	WEIGHT	MORPHINE	NEMBUTAL	SCOPOLAMINE OR ATROPINE	
	lb.	gr.	gr.	gr.	gr.
Up to 2 mo.	7-10	1/480	.....	1/600	1/400
2-3 mo.	10-12	1/320	.....	1/600	1/400
3-4 mo.	12-14	1/240	.....	1/600	1/400
4-7 mo.	14-16	1/144	.....	1/600	1/400
7-11 mo.	16-19	1/112	.....	1/600	1/400
11-18 mo.	19-24	1/96	..1/4	1/450	1/300
18-24 mo.	24-27	1/72	..1/4	1/450	1/300
2-3 yr.	27-30	1/64	..1/4	1/400	1/300
3-5 yr.	30-40	1/48	..1/2	1/300	1/200
5-8 yr.	40-55	1/32	..1/2	1/300	1/200
8-10 yr.	55-65	1/24	1	1/200	1/100
10-12 yr.	65-80	1/16	1	1/200	1/100
12-14 yr.	80-90	1/8	1 1/2	1/150	1/75
Adult	90 plus	1/8-1/4	1 1/2	1/150-1/100	1/75

Inspection of the doses of morphine given will reveal that each is an even division of an 1/8 gr. hypodermic tablet. And here a word of warning is necessary. The average general hospital seldom is called upon to prepare the minute doses which are employed for infants. Unless this is done with great accuracy, the patient will be either oversedated or undersedated. For example, consider the preparation of a hypodermic containing gr. 1/480 of morphine sulfate from the gr. 1/8 hypodermic tablet. The method usually tried seems to be

solved in 30 cc. sterile distilled water and that 0.5 cc. be used for the preoperative medication. All of our preoperative morphine doses are calculated so that the amount ordered will be in 0.5 cc. of solution. Similarly with scopolamine, a 1/200 gr. tablet is dissolved in 1.5 cc. of solution, and 0.5 cc., containing 1/600 gr. scopolamine hydrobromide, is given. In many hospitals where nurses are not accustomed to preparing these small doses, considerable inaccuracy results from failure to observe these essential details.

Although preoperative medication can be given by mouth, the results from this route of administration are totally unreliable. Varying amounts of the drug will be absorbed in widely differing periods of time. Therefore, these drugs are best administered by hypodermic injection. Much can be done to relieve the discomfort of a hypodermic injection. The skin is prepared with alcohol, which is allowed to dry before inserting the needle. Alcohol will seep in around the needle puncture and cause considerable unnecessary b u r n i n g pain. Needles, which should be sharp and free from barbs, are sterilized by boiling or autoclaving, since residual alcohol in the needle from chemical sterilization also produces pain.

No child enjoys a hypodermic injection, and the majority will fight against it. It is probably wise to use a Luer-Lok syringe, so that during struggling the chances of spilling the medication by separation of needle and syringe are minimized. Once the needle is inserted, the medication should be injected rapidly and the needle removed at once.

Attention to details involved in preparation of the preoperative hypodermic medication for infants and children is essential for accuracy of dose and reliability of response. It is the lack of attention to these very details by nursing personnel that has resulted in the unpredictable response to such medication and has fostered the conclusion that preanesthetic medication is unsafe and unsuited to very young patients. Many a child has been denied the advantages of preoperative medication because of some unfortunate experience with an overdose, when the real fault lay in carelessness or an indifferent technic. On the patient's record, in the nurse's daily notes, the steps taken to prepare the hypodermic should be given in full, so that there can be no question concerning the dose that the patient received.

Barbiturates are given either orally or rectally. Usually the oral route is productive of more reliable results. The drug can be given in Karo or Cartose, either of which minimizes the extreme bitterness of the drug and is far more palatable than the commercial elixirs.

The combination of morphine and scopolamine with or without barbiturates is given as preanesthetic medication one hour before the scheduled time for anesthesia. Since operating time rarely f o l l o w s the expected schedule, a certain leeway is allowed. Such premedication is usually effective for periods of forty-five minutes to one and three-fourths hours. If a longer period of waiting is necessary, the scopolamine should be given again in a dose equivalent to one-half the original amount. Atropine, when used, should be given separately, one-half hour before anesthesia to produce the desired results.

Children over 6 or 7 years of age are often helped by receiving a sedative dose of nembutal the night before operation. This assures a restful sleep, and its

(*Continued on page* 305)

CHOICE OF ANESTHESIA IN OBSTETRICS

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The purpose of this article is to discuss certain factors which, in my opinion, have not been accorded due importance in the choice of anesthesia in obstetrics. Physiologic changes brought about by pregnancy and conditions inherent in the gravid state impose difficulties in anesthesia not encountered in any other branch of medicine. In obstetric operations the anesthetist has the dual responsibility of attending the needs of both mother and child, a condition that does not exist in any other field of medicine and one that complicates and doubles the risk of any method of relieving pain.

Most surgical operations are planned in advance so that they may be done under the best possible conditions. This enables the anesthetist and surgeon working together as a team to plan for the proper preparation of the patient, the proper use of drugs preliminary to anesthesia, and the proper choice of anesthetic for the operation to be performed. In obstetrics this is seldom possible. The onset of labor usually cannot be predicted, nor can the character of the labor be foretold with any great degree of accuracy. The result is that no definite plan of action can be charted before labor starts. I think it is important at this point to stress the fact that one should have no "routine" method of handling obstetric cases. To do so would be to attempt to fit the patient to the procedure rather than logically to select the best procedure to meet the conditions actually present. It is therefore unwise to promise any patient before labor starts that a specific type or method of anesthesia will be used.

The pain of labor may be minimized by drugs that produce amnesia, analgesia, and anesthesia. These are separate and usually distinct effects, and one must not be confused with another. Amnesia is the loss of memory; in connection with our discussion it is the loss of memory of pain. Analgesia means the loss of the sensation of pain. Anesthesia signifies complete loss of all modalities of sensation. It therefore implies at least temporary paralysis of the sensory system. Anesthesia may or may not be associated with unconsciousness. Unconsciousness itself does not necessarily imply that anesthesia exists. No one drug is as yet available which in varying and safe dosages will

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produce all three conditions, i.e., amnesia, analgesia, and anesthesia. Some drugs will produce amnesia when administered in well tolerated dosages but will produce analgesia only when given in dangerously large amounts and anesthesia only when given in toxic doses. It is therefore of primary importance to know the pharmacologic action, effects, and dosage of the drugs to be employed. It is also extremely important to know the toxic effects of any drug and to have available and know how to use the antidotes for the unusual or toxic reactions of the drug.

Leaders in the medical profession have stated repeatedly that in the present state of our knowledge completely painless labor cannot be attained with safety to mother and child. Let us, therefore, not delude either our patients or ourselves by thinking that any drug or combination of drugs or any method or combination of methods now known will effect painless labor with complete safety to mother and child. To promise painless labor and then to attempt to fulfil this promise will inevitably lead to disaster. The safety of any method for relieving pain in labor must be measured by the bad results obtained, not by the number of cases in which it has been used without apparent harm. In our discussion today let us agree that we are considering only such relief from pain in labor as can be attained with safety to both the mother and the fetus. Motherhood entails many sacrifices, one of the first of which is the willingness to suffer a reasonable amount of pain in labor in the interest of her child's welfare. When the situation is explained in this way, almost all women are willing to accept a fair share of the discomforts and dangers of childbirth. Proper psychologic preparation of the patient for labor is obviously of great importance, and the time so spent in preparing patients pays big dividends in co-operation of the patient and smoothness of labor.

Relief of pain in labor may be divided into two parts: (1) methods of minimizing pain in the first stage and early second stage of labor and (2) methods of minimizing or eliminating pain at the time of actual delivery. In the first instance drugs and methods of producing amnesia and at least partial analgesia are utilized. Such methods seem to run in cycles, and I have lived through a number of such cycles in my professional career. The first such method in my experience was the use of morphine sulfate and scopolamine hydrobromide to produce so-called twilight sleep. This was followed by the Gwathmey method of rectal analgesia using morphine sulfate, scopolamine hydrobromide, and the rectal instillation of ether, quinine sulfate, and alcohol and oil. Next came the barbiturates, amytal, nembutal, pentobarbital, seconal, and so forth. These in turn were supplanted in popular favor by paraldehyde for a brief period and then by demerol and scopolamine hydrobromide and at about the same time by continuous caudal analgesia. Oddly enough, each method attained

widespread acceptance shortly after its introduction and was hailed by its proponents as the ideal means of relieving pain in labor. Then came reports of unfavorable reactions and fatalities to babies and mothers, followed by a loss of popularity, and finally each method fitted into its proper niche in obstetric practice. All of these methods are used today with good results when used in moderation and in properly selected cases. Each method has its good points, but unfortunately all have definite disadvantages and limitations so that none can be considered ideal.

An ideal method of analgesia in labor must meet the following minimal standards: (1) safety for both mother and child, (2) no interference with the progress of labor, (3) no depression of respiration in the fetus, (4) no increased bleeding in the third stage of labor, (5) adequate analgesia for a sufficiently long time, (6) simplicity and safety of administration, (7) reliability of action, (8) freedom from undesirable side effects, (9) economy, and (10) ready availability.

These criteria clearly expose the shortcomings of the analgesic methods so far devised. Morphine and scopolamine interfere with voluntary expulsive efforts, thus increasing the incidence of operative delivery, and depress fetal circulation. Rectal analgesia prolongs the second stage of labor, is cumbersome and time consuming in administration, and frequently causes considerable rectal irritation and proctitis. The barbiturates are primarily hypnotics and antispasmodics and produce amnesia and analgesia only when administered in toxic doses. They frequently produce pronounced restlessness and excitation, and thus constant supervision of the patient is required. All barbiturates affect the respiratory tract and are contraindicated in diseases of the respiratory organs. Paraldehyde by rectum is too evanescent in action, irritating to the rectum, and productive of unpleasant side effects. Continuous caudal anesthesia is too time consuming and expensive, technically too difficult, potentially too dangerous, and pharmacologically too uncertain for general use in obstetrics. Its use should be restricted to carefully selected cases in well organized maternity hospitals under the personal supervision of obstetricians well trained in its administration and thoroughly familiar with the complications that may arise from its use. Demerol and scopolamine are presently enjoying widespread favor. These drugs produce minimal unfavorable reactions, have a wide margin of safety for both mother and child, and generally give quite satisfactory relief from pain. They do increase the incidence of operative deliveries and probably somewhat increase postpartum bleeding. Sister Peter's paper will elaborate on these various methods of relieving pain in labor. With the single exception of continuous caudal analgesia, none of these methods gives sufficient anesthesia for actual delivery, and hence all must be supplemented in the late second stage of labor.

The same drugs are employed for anesthesia for delivery as are used in any other branch of surgery. For obstetric purposes they may be administered by inhalation, rectal instillation, intravenously, by spinal injection, or by local infiltration. The choice of the method and of the particular drug to be employed in any case depends upon a number of factors, only a few of which will be discussed in this article.

Neither the ideal anesthetic agent nor the ideal method of administration is available at this time. Each drug and each method has its own inherent advantages and disadvantages. Therefore, the problem in anesthesiology is to select the best drug and the best method of its administration for all the conditions present in any particular obstetric case. In a discussion of this sort only broad general statements can be made. Specific details must be left to the knowledge, training, skill, and judgment of the operating team, and by the operating team I mean not only the anesthetist and the obstetrician but all the personnel in the delivery room.

In my opinion the operating team personnel is a most important factor in the choice of an anesthetic. The skill, training, and experience of the anesthetist, the technical skill, speed, and mental equilibrium of the obstetrician, and the training and good common sense of the other persons in the delivery room are frequently the chief determining factors in the selection of an anesthetic.

When a specially trained medical anesthetist is available, the choice of anesthetic should always be left to him. Under such conditions the obstetrician may be certain that the decision will be made on the basis of full consideration of all factors in the case. However, at this time few such anesthetists are available. Most hospitals still depend upon nurse anesthetists. I believe this is entirely satisfactory provided real teamwork exists between the anesthetist and the obstetrician, and no attempt is made to exceed the limitations of the training, skill, and ability of either one. For example, it would be unwise for a physician to insist on using spinal anesthesia if his anesthetist is unfamiliar with this method. Although the surgeon might administer the anesthetic skilfully, he could not properly perform all of the duties of both anesthetist and surgeon throughout the course of the operation, and a complication under such conditions could prove disastrous. On the other hand, an anesthetist who is partial to the intravenous use of pentothal sodium ought not persuade a slow operator to attempt cesarean section with this drug as the anesthetic. Death of the baby might result. A nurse anesthetist who experiences difficulty with venipuncture ought not be asked to use pentothal sodium intravenously, for painful induration or extensive slough may occur at the site of injection. Serious difficulties may arise in the course of an operation done under an anesthetic that leaves the patient conscious, by a surgeon who loses his self control and "blows

up" under unusual stress and strain or from indiscreet remarks made by anyone in the operating room. Similiar examples could be given almost without limit, but these suffice to illustrate the point that these bad results occurred because the anesthetic was poorly chosen for the particular operating team, and that excellent results could have been obtained by the same team using another anesthetic.

The patient herself is another important factor in the choice of anesthetic. I do not refer to such obvious complications as pulmonary, cardiac, or renal abnormalities, but rather to the mental and nervous characteristics of the patient. Not infrequently a patient expresses a desire to receive an anesthetic that will allow her to retain consciousness at the time of delivery. Provided the patient is temperamentally fitted for this, it may be well to grant such a request and use spinal, caudal, or local infiltration anesthesia for delivery. In other cases, however, these types of anesthesia are very unsatisfactory because the patient is high strung, excitable, or plain neurotic, and hence too susceptible to external stimuli. Such a patient does much better under an anesthetic that produces unconsciousness.

The volume of work and the number of nurses available in the delivery room should also be considered in the selection of an anesthetic. Spinal, saddle block, caudal, or local infiltration anesthesia requires more time from the obstetrician and nursing staff than does an inhalation anesthetic. Furthermore, for best results these forms of anesthesia require silence and complete absence of hustle and bustle in the delivery room. When labor rooms and delivery rooms are not separated by soundproof partitions and several patients are in active labor with nurses and doctors milling around, only the most phlegmatic patient can undergo delivery with tranquillity while conscious. Most maternity hospitals have been and are still operating far beyond the capacity for which they were built. The nursing staff either has been increased to the point where the nurses actually get in each other's way, or, more commonly, has been kept at the same complement as when the daily patient census was considerably lower, and hence is much overworked. Either condition leads to noise and confusion and definitely restricts the choice of anesthesia. In my opinion, it is unfair to employ time-consuming procedures in the care of one patient to the detriment of other patients when simpler, quicker methods will give equally good results. We all wish occasionally to employ some of the less frequently used forms of anesthesia, but we should do so only when the delivery room staff is not needed for more urgent duties.

What drug and what method of administration, then, should be employed in uncomplicated obstetric cases under presently existing conditions? Without doubt, inhalation anesthesia using nitrous oxide, ether, ethylene, or cyclopropane, in this order of preference, will give the best results in most cases. Nitrous oxide-oxygen anesthesia

is particularly effective for patients who have had barbiturate, morphine, or demerol analgesia because it acts synergistically with these drugs. It is nontoxic, has a wide margin of safety for both mother and fetus, does not suppress fetal respiration, and is quick acting, pleasant to take, and easy to administer; recovery from its effects is also rapid. Nitrous oxide-oxygen anesthesia together with open drop ether must be considered the cornerstone of obstetric anesthesia under average conditions.

Ethyl ether is a very satisfactory anesthetic agent. It gives good muscular relaxation, has a wide margin of safety, is administered with a minimum of apparatus, and causes minimal organic damage. Contrary to generally accepted belief, ether is a good anesthetic in the presence of cardiac disease and is not specifically contraindicated in the presence of disease of the respiratory tract, although in such cases other forms of anesthesia are usually more satisfactory. Its chief disadvantages are the long induction and recovery periods and its effect on the gastrointestinal tract. Ether is the inhalation anesthetic of choice except in individual cases in which some other potent inhalation agent can be demonstrated to have some specific advantage.

Divinyl ether, ethylene, and cyclopropane have only limited usefulness in obstetrics. Their disadvantages so far exceed their advantages that their use in obstetrics is seldom justified. Chloroform is seldom employed in obstetrics in this part of the United States.

Under present day conditions of practice, inhalation anesthesia must be looked upon as the foundation of obstetric anesthesia. All other forms of anesthesia are, by comparison, mere froth. In individual cases some other form of anesthesia may offer definite, specific advantages and should therefore be used in preference to inhalation anesthesia, but such cases are the exception, not the rule.

The intravenous use of pentothal sodium as an anesthetic agent in obstetrics is seldom indicated. Its use must be restricted to procedures that can be performed in a short time. Actually, the condition resulting from the intravenous injection of pentothal sodium or evipal is acute barbiturate poisoning rather than true anesthesia and differs from the poisoning caused by other barbiturates only in the shorter duration of its action. The concentration of pentothal sodium in the fetus reaches the same level as that in the mother in seven to ten minutes. It is important, therefore, that all preliminaries to actual delivery be completed before pentothal sodium is administered, so that the fetus may be delivered before a dangerously high concentration of the drug is reached in the fetal blood.

Spinal anesthesia is the most dangerous of all anesthetic methods used in obstetrics. This is still true in spite of improvements recently made in its use. The use of smaller doses of the anesthetic agent, continuous rather than single injection administration, the use of hyperbaric solutions which are more

controllable, and confinement of the level of anesthesia to lower regions of the body have all contributed to the safety of spinal anesthesia, but the method still suffers by comparison with almost any other anesthetic. Its use in obstetrics is therefore unwarranted in the average case and should be restricted to the complicated case in which inhalation anesthesia is contraindicated and in which spinal anesthesia offers specific advantages over other forms of anesthesia. In many such cases local infiltration will serve very satisfactorily and be definitely safer.

Local infiltration anesthesia in obstetrics is slowly increasing in popularity. It should be more extensively used. Contrary to the belief of many obstetricians the technic of local infiltration is simple. It has absolutely no harmful effect on the fetus, and to date no maternal death attributable to direct infiltration anesthesia has been reported. This cannot be said of any other type of anesthesia. There are no medical contraindications to the use of local anesthesia. Its chief disadvantage is its short duration, which limits its use to the late second stage of labor when delivery is imminent.

SUMMARY

Many factors must be considered by the anesthetist and obstetrician working together as a team before an anesthetic is selected for delivery. The safety of mother and child is the factor of primary importance and to it all other considerations must be subordinated. The ideal anesthetic for all obstetric cases is not yet available.

Ether and nitrous oxide-oxygen are the best anesthetic agents under ordinary conditions for uncomplicated obstetric cases and for complicated cases in which inhalation anesthesia is not contraindicated. Local infiltration anesthesia deserves more widespread use. Rectal, intravenous, spinal, and caudal anesthesia should be reserved for selected cases in which the method of choice offers specific advantages over any other type of anesthesia.

MODERN METHODS OF MINIMIZING PAIN DURING LABOR AND CHILDBIRTH

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The ability to tolerate pain seems to vary in inverse ratio to the education of the mother of today. Years ago, physicians and midwives delivered girls who accepted a long hard labor with an inbred Stoicism. The attitude toward labor of the expectant mother of yesterday is vastly different from that of the expectant mother today. Then pain was accepted as an unavoidable part of labor; today we are searching for analgesic agents that will relieve pain and afford a feeling of restfulness and relaxation to the mother without endangering her or the child.

"Painless childbirth" is a misnomer. Regardless of how remarkable the pain-alleviating agent may be, no one drug has yet been proved perfect. For the protection of the mother and child, several aspects of any obstetric analgesic must be considered. Does it retard the progress of labor, increase the danger of hemorrhage, or prevent obstetric intervention? Does the drug have any effect on the fetus in utero? Does it retard respiration of the baby or have any effect upon the newborn during the first few days of life? All of these interrogations influence any evaluation of the value, uses, and usefulness of any agent now recommended for obstetric analgesia and anesthesia.

The ideal for which we strive is the reduction of pain during the entire period of labor. At the Fitzgerald-Mercy Hospital we have attempted to accomplish this in the first and in part of the second stage of labor by the use of narcotics and barbiturates in combination with scopolamine. The patient co-operates fully because today the expectant mother is familiar with the term "painless childbirth." No analgesic is given until labor has actually begun. The patient must first be prepared mentally. Sedation is given when the pains are well established and dilatation of the cervix has progressed. The choice of drug and the dosage depend upon whether the patient is a primipara or multipara. For primiparas, analgesia is started when the head is distinctly engaged, pains are well established, the cervix is half effaced and preferably somewhat dilated, and the patient needs a sedative. An analgesic is given to multiparas as soon as we are sure labor is definitely in progress. Analgesia will definitely retard labor if the drug is given too soon, regardless of the combination employed.

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For an initial injection, we use demerol, 100 mg., and scopolamine, gr. 1/100, in combination with one of the barbiturates, gr. 3 to 4 1/2. In one hour scopolamine, gr. 1/200, is administered; if additional sedation is required, demerol, 50 mg., may be given. Scopolamine has been found to be the most useful agent for producing amnesia. This drug in combination with demerol and one of the barbiturates has so far proved a satisfactory analgesic for both primiparas and multiparas.[1,2] In our experience this combination has produced no respiratory depression in full term or premature infants even when it has been administered shortly before delivery. In cases of persistent occiput posterior presentation or for patients who are subject to long labor, morphine sulfate, gr. 1/6, may be administered as the initial dose, followed in two hours by demerol, scopolamine, and one of the barbiturates in the aforementioned dosage.

If the patient becomes restless, scopolamine, gr. 1/200, may be given every two hours until the patient sleeps between pains and is roused only by uterine contractions. As labor progresses, the cervix becomes completely dilated, and the presenting part is evident. The patient is now ready for anesthesia for the expulsion of the fetus. In our institution, an inhalation anesthetic is most often used. A mixture of nitrous oxide, 85 per cent, and oxygen, 15 per cent, has been proved by experience to be the best combination for a forceps delivery, breech presentation, or for other cases in which obstetric intervention may be necessary. Ether vapor may be added to the aforementioned mixture. With this combination, pain is completely abolished, relaxation is obtained, and there is a wide margin of safety because of the easy administration of oxygen. Of course, it should not be inferred that this mixture is used routinely, for in every case, the patient's physical and mental condition is taken into consideration. There should be no question of anoxia since the flow of oxygen is readily controlled by the anesthetist, and even pure oxygen may be given if necessary. In fact, it is our custom, after the delivery of the child and before the cord is cut, to flood the patient's respiratory system with pure oxygen. This has a beneficial effect on the baby, as is evidenced by a spontaneous cry, disappearance of cyanosis, and a healthy pink color of the skin. In none of our cases have we had an immediate or a late reaction to the anesthetic. With this type of anesthesia, operative procedures such as versions, episiotomies, and forceps deliveries can be satisfactorily carried out. An episiotomy can be quickly and easily repaired, and the mother recovers promptly from the anesthesia with little discomfort.

A patient who is admitted to the hospital in active labor is usually given a single injection of demerol and scopolamine and then nitrous oxide and oxygen

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1. Barnes, Josephine: Pethidine in labor: Results in 500 cases. Brit. M. J. 1: 437, April 5, 1947.
2. Flatt, W. D.: Relief of pain in labor with demerol. Canad. M. A. J. 55:43, July 1946.

with each pain until she is ready for delivery. Best results are obtained when the gas is given just before the uterine contraction becomes painful.

In the few cases in which nitrous oxide is contraindicated, we find ether to be the best general anesthetic because of its wide margin of safety.

In 1947, 2,200 mothers went home from our hospital with apparently normal, healthy babies. Total deliveries numbered 2,280. Forty-eight babies were stillborn as a result of cord strangulation or other abnormality. There were 32 neonatal deaths and 1 maternal death. The maternal death was of a 32 year old primigravida, six months' pregnant, who died undelivered from paroxysmal auricular tachycardia. The causes of the neonatal deaths were: prematurity (14 cases), congenital cardiac abnormality (4), atelectasis (5), erythroblastosis foetalis (2), cerebral hemorrhage (2), fetal hydrops (2), and intracranial hemorrhage (2).

There has been an increase in the number of deliveries under spinal anesthesia.[3] For a while we were dubious as to the usefulness of this method of anesthesia in obstetrics, owing to the exaggerated criticism of it, not only by the laity but by the profession at large. Now we find ourselves being converted. Procaine crystals, dissolved in 1.5-2 cc. of spinal fluid and injected into the third or fourth lumbar interspace, is the spinal anesthetic of choice. We usually use a small dose, 50-65 mg., which provides sufficient anesthesia for a painless delivery, with good perineal relaxation, and allows ample time for a forceps delivery and repair of an episiotomy. Outlet forceps are usually necessary, because under spinal anesthesia the expulsive efforts of the patient are weakened. In cases in which the obstetrician may require increased uterine relaxation, such as a difficult forceps delivery or a retained placenta, a little general anesthesia may be necessary. Spinal anesthesia is of special value in the delivery of premature babies. The amount of bleeding during the actual delivery is remarkably small. Up to the present time, we have not used spinal anesthesia very often for vaginal deliveries, but it has been used in 75 per cent of the cases of cesarean section with successful results.[4,5]

Saddle block anesthesia differs from spinal anesthesia in that it is subject to greater control. Saddle block anesthesia is confined to the perineal area and is produced by injecting small doses of the spinal anesthetic into the subarachnoid space with the patient in the sitting position. By using drugs of prolonged action, such as pontocaine or nupercaine, sufficient anesthesia may be obtained by a single injection. Although we have not had too much experi-

3. Weaver, R. F.; Adamson, D. L., and Johnson, F. L.: Spinal anesthesia in vaginal delivery: A report of 1,542 cases. Am. J. Obst. & Gynec. **51**: 764, June 1946.

4. Ullery, John C.: Continuous spinal analgesia in cesarean section: Its advantages, technique and an analysis of three hundred cases. Am. J. Obst. & Gynec. **52**: 100, July 1946.

5. Heard, Kenneth M.: Spinal anesthesia —the protector of the baby in cesarean sections. Anesth. & Analg. **25**:191, Sept.-Oct. 1946.

ence with this type of anesthesia, its possibilities deserve mention.[6]

Likewise, pudendal block has been rarely used in our hospital.[7] In the few instances in which it was used, it was successful. It is a form of local anesthesia produced by blocking the pudendal nerves with a series of injections of 1 per cent novocain. Local anesthesia has many advantages over other forms of anesthesia in obstetrics.[8] No deaths have resulted from its use, and pulmonary complications do not occur. Moreover, there are no harmful effects on the fetus. In rare instances drug shock may occur. The accidental injection of the drug directly into a vein may cause a serious complication. Any other complication is rare.

A new method of combining pentothal sodium and nitrous oxide and oxygen has been introduced in obstetric anesthesia.[9] For over two years it has been used with good results. The pentothal sodium given by rectal instillation is used as a basal anesthetic and has an analgesic effect. No other barbiturate or opiate is given. Atropine, gr. 1/150, is then given by subcutaneous injection. Nitrous oxide is administered for the delivery and for repair of the episiotomy. The pentothal sodium is given in a dose of 0.01 Gm. per Kg. body weight. The safety of this combination is still questionable, and the method has not yet been accepted by those of us who have had little experience with it.

6. Parmley, Ray F., and Adriani, John: Saddle block anesthesia with nupercaine in obstetrics. Am. J. Obst. & Gynec. **52**:636, Oct. 1946

7. Eisaman, Joseph R., and McHenry, Thomas, III: Pudendal nerve block in obstetrics (perineal field block). Pennsylvania M. J. **49**: 964, June 1946.

8. Greenhill, J. P.: Anesthesia in obstetrics. Am. J. Obst. & Gynec. **54**:74, July 1947.

9. Pentothal anesthesia for cesarean section, Queries and Minor Notes. J.A.M.A. **134**:968, July 12, 1947.

SUMMARY

The goal of the anesthetist in obstetrics is the reduction of pain during the entire period of labor. No drug has yet proved perfect, and the selection of the drug is based primarily on its safety to mother and child. The range of methods from which a selection may be made and our preferences have been discussed.

PREANESTHETIC MEDICATION

(*Continued from page 294*)

residual effects the morning of operation may be valuable in aiding the child to accept the anesthesia with greater calm.

Finally, if regional anesthesia has been chosen for a young child, the sedation recommended in this table is inadequate. In most instances satisfactory results will be obtained by doubling the amount of morphine. Barbiturates are valuable as they provide considerable protection against the toxic effects of local anesthetic drugs in addition to their sedative action. Scopolamine also should be given, so that when supplementary general anesthesia becomes necessary, it can be done easily and safely.

SUMMARY

The need for preanesthetic medication for the pediatric patient and the advantages and the disadvantages have been discussed. The advantages far outweigh the disadvantages.

ANESTHESIA FOR THORACIC SURGERY

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Of the various methods of anesthesia used for all types of operative procedures, the only really practical one for thoracic surgery is a form of inhalation anesthesia given by the intratracheal route. This provides the necessary narcosis without undue depression. It gives ready access to the tracheobronchial tree with a suction catheter for the removal of secretions, pus, and blood. It gives complete control of the lungs, which can be inflated and deflated at will through the always patent airway provided by the intratracheal catheter.

Anesthetic Agents

Several gaseous agents may be used. Nitrous oxide is not suitable because a sufficient depth of anesthesia cannot be obtained without danger of anoxia. These patients, especially, should receive the highest possible concentration of oxygen, since even a brief period of anoxia may start a chain of events leading to disaster. One suggested technic involves the combined use of curare and nitrous oxide. The curare is used to eliminate the respiratory effort while the patient is anesthetized with nitrous oxide and oxygen after receiving heavy premedication. After intubation the anesthesia is maintained with the apneic technic by periodic inflation of the lungs by manual compression of the rebreathing bag. This eliminates voluntary diaphragmatic excursions and provides a motionless operative field. The technic has several disadvantages. The danger of anoxia is always present when nitrous oxide is used to keep a patient anesthetized, especially in the presence of an open pneumothorax. The large doses of curare necessary to maintain the apnea may lead to circulatory collapse from loss of muscular tone and loss of respiratory effort to help the venous return to the heart.

Ethylene may be discarded on the same basis as nitrous oxide, in that it does not provide good anesthesia when used with a sufficient amount of oxygen to prevent anoxia.

Cyclopropane and ether are the two suitable agents, and either or both are used in most clinics. They are both potent enough to provide any desired depth of anesthesia while allowing a more than adequate concentration of oxygen to be delivered to the patient.

Cyclopropane has several good features. It is pleasant as an induction agent, is rapid in action,

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and has a relatively short recovery period. But it also has definite disadvantages. It has a direct depressant effect on respiration, the amplitude of respirations becoming less as the depth of anesthesia is increased. This diminishes the oxygen intake in a patient who can ill afford any reduction in oxygenation. It is a parasympathomimetic drug, which is undesirable when the surgeon is working near the vagus nerve and is constantly stimulating it. Traction on the hilus in some instances may cause marked cardiac arrhythmia, severe respiratory depression, or apnea.

Ether has several advantages over cyclopropane as the agent of choice for anesthesia for intrathoracic surgery. It is easier to maintain longer an even level of anesthesia which will not quickly lighten and cause the patient to attempt to cough when anesthesia is temporarily discontinued for the use of suction. Irregularities in pulse and respiration are infrequent, and there are usually no changes in cardiac rhythm when the hilus or vagus is stimulated. Ether is a sympathomimetic drug, tending to counteract the effects of vagal stimulation. Respirations are not depressed but rather are stimulated and remain full and deep. Slight obstruction becomes apparent, whereas with cyclopropane the obstruction may become severe before it is recognized. The oxygen concentration with ether is slightly higher than with cyclopropane, although when the concentration of oxygen is approximately 90 per cent, a 5 per cent difference one way or the other is not very important.

The disadvantages of ether are that the recovery period is longer and the incidence of nausea and vomiting greater. These are outweighed by the advantages obtained during the operative procedure.

For these reasons a combination of both agents has been found useful. In this series of cases, cyclopropane-oxygen was used for induction, and ether added and used for maintenance after the cyclopropane was discontinued.

It has been suggested that pentothal sodium be used for induction and intubation be performed while the paitent is under its effect. With this method induction is rapid, and the electrocautery may be used while the chest is being opened. After the pleura is opened, anesthesia is maintained with an ether-oxygen mixture. This technic should be discarded because of its dangers. Attempts to perform intubation on a patient anesthetized with pentothal sodium may result in severe laryngospasm with resultant anoxia. Pentothal sodium is also a direct respiratory depressant. It may cause anoxia from diminished respiratory exchange.

The apneic technic with cyclopropane has been widely discussed. In thoracic surgery it is desirable for the surgeon since it provides a completely quiet operative field while he is working inside the chest. However, there is the danger of unnoticed secretions. While the lungs are being manually inflated by the anesthetist, secretions from the lung being operated on may pour into

the underlying lung which is carrying on the major part of respiration. The presence of these secretions may go unnoticed until they produce cyanosis. By allowing the patient to breathe spontaneously, the presence of secretions can easily be noted by signs of respiratory tract obstruction. Another bad feature of this technic is that the secretions may be forced into the periphery of the lung where they cannot be readily aspirated. This might be overcome by suction at regular intervals, but this is a haphazard method, relying more on a stated time interval than on the appearance of signs and symptoms. I believe that, on the whole, it is better to allow the patient to breathe spontaneously than to take over his respiratory effort.

Technic

The results of the anesthetic technic described were favorable in fifty consecutive cases in which Dr. H. C. Maier did the operation while I gave the anesthetic. The operations included pulmonary resection for carcinoma, abscess, and tuberculosis; removal of mediastinal tumors; and correction of cardiac abnormalities. The patients ranged in age from 3 to 70 years. There were no deaths.

The first step in anesthetic management is to visit the patient preoperatively to evaluate him and judge the amount of medication needed to have him in a suitable frame of mind for the operation. Premedication consists of morphine sulfate, gr. 1/4 to 1/8, and atropine sulfate, gr. 1/100 to 1/200, in the ratio of 25:1, given subcutaneously forty-five minutes to one hour before the start of the anesthesia. When ordering premedication it is important to keep in mind that the anesthesia is started about one-half hour before the time scheduled for operation and to allow for this when writing the time of administration. Otherwise, the medication will be given too near the start of anesthesia and will hinder rather than help the induction. In thoracic operations it is extremely important to avoid respiratory depression, and therefore the dosage must be individualized. To start with respiratory depression is to start with a great handicap. It is better to give too little premedication than too much.

The induction of anesthesia may be carried out either in an anesthesia room or in the operating room. The place does not make much difference if means for suction are available for use when the anesthesia is started. Patients with intrapulmonary lesions often have large amounts of secretions, and the anesthetist must be ready to aspirate them at a moment's notice.

The make of anesthesia machine used is of no importance if it is in good condition, with no leaks, and is equipped with a rebreathing system. Either the to-and-fro or the circle type may be used, depending upon individual preference.

The anesthetist must not allow himself to be rushed by the surgeon or the operating room nurses. As a result induction of anesthesia or intubation may be hurried. If the anesthesia is too

light when intubation is attempted, reflexes may be stimulated with resultant laryngospasm. Anoxia occurs, and the course of anesthesia is made difficult. Any period of anoxia, no matter how brief, will make the patient less tolerant to prolonged anesthesia.

Induction of anesthesia is carried out with a mixture of cyclopropane and oxygen. As soon as the patient has reached the lower first plane of the third stage, a pharyngeal airway is inserted and ether is added to the mixture at a steadily increasing rate and the flow of cyclopropane gradually diminished until the anesthesia is being maintained with ether. The level of anesthesia is then carried to the lower second plane and intubation performed.

The necessary sterile equipment is available on a special table to the right of the anesthetist. The mask is disconnected, the pharyngeal airway removed, and laryngoscopy performed. The intratracheal tube is inserted between the vocal cords far enough so that the inflatable cuff near the end of the tube just passes the vocal cords. Before the laryngoscope is removed, a small roll of bandage or a bite-block is placed between the teeth to prevent the patient from biting down on the tube. During this procedure and until the tube is secured with strips of adhesive tape, care is taken to keep the tube in the same place so that it does not slip in too far or slip out from between the vocal cords. The tube is then connected to the anesthesia machine and the plane of anesthesia stabilized. Infrequently during intubation the anesthesia becomes too light, and the patient attempts to cough or buck. After the level of anesthesia is deepened and the patient has reached a state of equilibrium, the inflatable cuff is blown up and the system tested to make sure it is airtight. It is always a good policy to test the cuff before inserting the tube to make sure the balloon has no leaks.

When the anesthesia is proceeding smoothly again, aspiration should be performed. The oropharynx should be cleaned out both through the nose and through the mouth. Then a catheter is inserted into the tube and aspiration performed. Thus secretions that may have accumulated are removed, and the anesthetist is assured that the catheter will pass easily in the future. The system is then connected up again, and when the anesthesia is well stabilized, the patient can be turned and placed in position for surgery. We usually use the lateral position, with the side of the lesion uppermost. The patient is securely positioned, the area prepared and draped, and the operation started.

The anesthesia is maintained in the upper second plane of the third stage for the operative procedure. This is deep enough for the patient to tolerate the presence of the tube in the trachea. The anesthesia is never allowed to deepen beyond the second plane. In this way the patient is not subjected to the depression associated with deep inhalation anesthesia over a long period. There is very little change in his status during the

whole procedure, even though it may last from six to eight hours.

The best indication of the depth of anesthesia is the size of the pupils. In thoracic surgery the character of respirations, which is the sign usually used in other types of operations, is not very reliable because it is distorted by the operative procedure.

During an operation in which the whole lung is not being resected but in which only a lobe or a mediastinal tumor is being removed, the lung on the open side of the chest is inflated every fifteen or twenty minutes for about one minute. This is extremely important for it prevents the lung from becoming edematous from too long a period of collapse. In a pneumonectomy this is not important, and the lung is usually allowed to remain collapsed during the whole procedure.

When closure is started, an attempt is made to lighten the anesthesia by emptying the rebreathing bag and flushing it with oxygen every two or three minutes. Under this regimen the patient has started to react, has active corneal reflexes, and resents the presence of the tube as evidenced by attempts to cough by the time closure is completed. Until the closure is airtight, the lung on the open side is kept inflated. When a lobectomy has been done, an underwater drainage system is set up and the lung inflated against this. When a pneumonectomy has been done, the chest is closed with a large catheter in place until the skin is sutured, to prevent tension pneumothorax. After closure has been completed, pressures are adjusted in the side from which the lung has been removed.

The intratracheal tube is left in place until the dressing has been applied and the patient rolled over onto the stretcher on his back and the pressure adjusted. The pharynx is thoroughly cleaned out through both the nose and the mouth. The suction catheter is then inserted through the tube until its end is an inch or two past the end of the tube, and they are withdrawn simultaneously. Any secretions that might have accumulated and been protected by the presence of the tube are now removed. A pharyngeal airway is inserted if the patient will tolerate it, the face mask of the anesthesia machine reapplied, and the patient given oxygen continuously while being taken to his room. The machine is disconnected only after the patient has been placed in an oxygen tent. Then, if nasal catheters are also to be used, they are inserted. In this way the patient is never deprived of the oxygen-rich atmosphere that he evidently needs. Exposing the patient to room air for only the short time necessary to transport him to his bed may result in profound collapse.

Discussion

The problem of partial or complete respiratory obstruction is of the utmost importance. It may occur either from the secretions of the patient or from some mechanical difficulty associated with the intratracheal apparatus. The proper handling of the situation is essential to the ultimate

successful conclusion of the operative procedure.

The most common diseases for which pulmonary resections are performed are bronchiectasis, carcinoma, chronic lung abscess, and tuberculosis. In each of these the expectoration of sputum or blood is a prominent symptom. As a result, obstruction may develop from the collection of these secretions in the lung not being operated upon when the patient is turned to the lateral position, when the pleura is opened, or at any time the diseased lung is manipulated by the surgeon.

The most important sign of respiratory tract obstruction is the tugging of the chin associated with the increased respiratory efforts and the use of the accessory muscles of respiration. The chin goes down with each inspiration, and concurrently diaphragmatic excursions are increased in depth and force. On rare occasions the presence of secretions is first indicated by a bubbling sound associated with respirations. Usually, their presence can be detected earlier by signs of obstruction. Cyanosis is a later sign of very serious import, and the obstruction should be corrected long before this develops.

The first step in the treatment is an attempt at prevention. In cases where excessive amounts of secretion may be expected, they can be removed to a large extent preoperatively by postural drainage and bronchoscopy. When trouble occurs during the operation, they must then be removed by means of a suction catheter passed down into the major bronchi through the intratracheal tube. The patient may be put in 20 degree Trendelenburg position to use the force of gravity as an aid. If, in spite of constant suctioning, the patient is still in distress from continuing obstruction and flow of secretions, the surgeon can help by modifying his procedure to tie off the bronchus first or by doing a tourniquet procedure.

The obstruction of a bronchus may promote anoxia in two ways. First, aeration is interfered with by the diminished area of lung surface available. Secondly, the increased respiratory efforts to draw more air into the lung result in a greatly increased intrathoracic negative pressure. This tends to draw fluid as a transudate from the tissues into the alveoli. The resultant pulmonary edema again interferes with oxygenation. This condition is recognized by contrasting the violence of the respiratory effort with the actual excursion of the rebreathing bag.

Since the suction catheter plays such an important role in the maintenance of a patent airway, it should be chosen with care. It should be small enough in diameter to pass easily through and beyond the intratracheal tube and not obstruct it. It must be soft, since it is passed beyond the end of the intratracheal tube and will rub against the mucosa of the trachea and the main bronchi. A stiff or rough tube may damage the mucosa and cause bleeding with resultant blood clot and complete respiratory tract obstruction. The length of the intratracheal

tube should be marked off on the catheter, so that one can tell when its tip has passed the end of the tube. A satisfactory catheter is an infant-size enema tube, which is of small caliber, soft, smooth, and has several holes in the end and sides. A glass connecting tip should be interposed in the rubber tubing connecting to the suction apparatus, so that the nature of the secretions being removed can be easily and quickly determined. During aspiration, the operative procedure must be interrupted, since stimulation of the trachea by the suction catheter and manipulations of the lung or hilus may precipitate severe cardiac arrhythmias or cardiac arrest from activation of the combined vagal reflexes.

Irregularities associated with respiratory reflexes are much commoner during cyclopropane anesthesia than during ether anesthesia, and for this reason ether is the anesthetic agent of choice. If the anesthetist must use cyclopropane, ether should be immediately added to the mixture upon the appearance of irregularities.

The intravenous administration of fluids may be handled by the anesthetist. It has always been my policy to do so, since intravenous therapy is most important in maintaining the patient in good condition. At first I used the ankle vein and usually prepared it with the cutdown technic. But it became impracticable, since every time the rate of flow needed adjustment, I had to leave the head of the table and run around to the foot where I got in the way of the scrub nurses and they in mine. The rate of flow was difficult to watch, since the apparatus was usually partially or completely covered with drapes. I have therefore adopted the policy of using the veins on the dorsum of the hand. In this way, I have the venoclysis right where it can be watched every minute. The usual procedure is to start with 200 cc. of saline and, then, before the pleura is opened, to add blood at the rate of 60-100 drops per minute for the rest of the operative procedure, usually from 500 to 1,500 cc. being needed during a pulmonary resection. This liberal use of blood is of great importance in keeping the patient in good condition. It is rare that the patient's blood pressure at the close of the operation varies more than 20 points from what it was preoperatively.

Summary

Co-operation and mutual confidence between the surgeon and anesthetist are of the utmost importance in thoracic surgery. The best results can be obtained only when they work together as team, realize the difficulties that each may encounter, and try to help each other to overcome them.

COMPLICATIONS DURING ANESTHESIA

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Of the many undesired reactions and complications which may occur during the administration of an anesthetic, those of major importance may be divided into four groups. These groups in the order in which they will be discussed in this article are: respiratory complications, circulatory complications, neurologic complications, and technical complications.

Respiratory Complications

Respiratory complications are the commonest and if not recognized and treated promptly may lead to asphyxia.

Anoxia is interference with adequate oxygenation of tissues. The causes are varied. The supply of oxygen may be inadequate owing to decreased oxygen concentration in the inspired air. Access of oxygen to the lungs may be impaired by obstruction of the airway. An overdose of a depressant drug may interfere with respiratory activity, as may restriction of thoracic and diaphragmatic movements as a result of restraint. Anoxia will also occur if the vital capacity is decreased owing to mechanical restrictions, such as pneumothorax and alteration of the pulmonary epithelium. Additional causes may be pulmonary edema and impairment of oxygen transport.

Whatever the cause, anoxia must be treated promptly. If treatment is delayed, death from circulatory failure may occur within a few minutes or a few hours, or, should the patient recover, symptoms of cerebral damage will be present.

Cyanosis is characterized by a bluish discoloration of the skin and mucous membranes due to an excess of reduced hemoglobin in the blood. The cause is a reduction in oxygen tension in the alveoli as a result of various conditions. The respiratory tract may be obstructed. The partial pressure of oxygen in the inhaled mixture may also be decreased. The tension in the alveoli may likewise be reduced by respiratory depression or failure, caused by interference with respiratory movements or a decreased vital capacity. The same result obtains when capillary circulation is reduced as it is in the presence of circulatory failure or when a vessel or an extremity is compressed.

Apnea is the cessation of respiratory movements. It may beproduced by hypocapnia deriving from hyperventilation, which raises the threshold of the respiratory center to carbon dioxide. Other causes of apnea are re-

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flex stimulation, overdosage of a depressant drug, laryngeal or bronchial spasm, obstruction of the airway, increased intracranial pressure or other neurologic disturbances, and circulatory failure.

Hyperpnea is an abnormally excessive rate or depth of respiration caused by excessive carbon dioxide or stimulation during light anesthesia.

Hypercapnia is due to excessive carbon dioxide. The blood pressure is increased with no change or only a slight increase in pulse rate. Respirations are depressed, and a gasping type of activity results in hyperpnea. Other signs and symptoms are twitching of the muscles followed by convulsions, increased depth of narcosis, cardiac arrhythmias, and noisy respirations. There are a number of reasons for the appearance of hypercapnia: The soda lime may be exhausted, the ratio of dead space to the tidal volume thus becoming out of proportion. The carbon dioxide flow meter may be on. Exhalation valves may be defective, or the filter shut off. The carbon dioxide cylinder may be attached to the wrong yoke. Also, oxygen and other gases may be contaminated with carbon dioxide.

Excessive secretions of mucus from salivary glands and other mucous membranes may sometimes lead to unpleasant incidents. This may occur if premedication is insufficient or not given at a proper time. Too high a concentration of the anesthetic drug, excitement or anoxia during induction, and the administration of parasympathetic stimulants or sympathetic depressants are other causes. To correct the condition, the patient's head should be lowered, aspiration instituted, bronchoscopy performed in severe cases, and atropine administered intravenously, as indicated.

Pulmonary edema is the transudation of fluids from the pulmonary capillaries to the alveoli. Among the conditions producing pulmonary edema is obstruction to inspiration from laryngospasm which causes excessive negative pressure in the alveoli. Toxic agents, such as nitric acid, an impurity of nitrous oxide, can cause an alteration in the pulmonary epithelium with the same result. Cardiac failure causing an increase in pulmonary venous pressure, changing the patient's position, improper technics in the administration of the anesthetic, and overburdening the circulatory system with an excess of fluids given intravenously are other causes of pulmonary edema. The condition is recognized by noisy respirations, or other signs of obstruction to respiration, and by the presence of a pinkish frothy secretion coming from the mouth.

Before pulmonary edema may be treated, the cause must be determined. Among the measures used to correct the condition are the administration of oxygen under pressure with bag and mask, phlebotomy or dehydration to reduce pulmonary venous pressure, and aspiration of the bronchi. The patient's head should be raised and the feet lowered. The application of tourniquets to increase peripheral venous stasis and the adminis-

tration of symptomatic treatment also may be indicated.

Obstruction of the airway is the commonest difficulty encountered during the administration of an anesthetic and if not corrected may result in anoxemia, carbon dioxide retention, or an inability to anesthetize the patient. Such obstruction may be due to relaxation of the tissues, such as the muscles of the neck, tongue, pharynx, or epiglottis, or to laryngospasm, partial or complete. One must always be aware of the possibility of obstruction from foreign bodies, such as false teeth, as well as from vomitus or blood clots. Mucus, saliva, or the accumulation of purulent material from abscesses are other possible causes. Not to be overlooked are anatomic abnormalities or defects, such as edema of the trachea, polyps, tonsils, or tumors. The artificial airway may be faulty. Likewise, there may be bronchospasm caused by asthma or vagal stimulation. Among the mechanical causes of obstruction are bad posture such as in the Trendelenburg position or hypoextension as well as hyperextension of the head, and the presence of restraining straps or bandages. Inhibition of thoracic movements with resultant obstruction may also result from assistants' leaning on the patient's chest. Noisy or labored respirations, an elevation of blood pressure, rapid pulse, and cyanosis are all signs of obstruction of the airway.

There are a number of methods of maintaining a patent airway. The patient's head may be supported on a small pillow with chin up; at the same time, the position of the patient should be checked to make certain that there is no interference with respiratory movements. The insertion of an artificial airway or an intratracheal catheter is insurance against obstruction. Likewise, aspiration should be used immediately to remove foreign bodies and secretions. If acute complete obstruction is not treated promptly, asphyxia and circulatory and respiratory failure will supervene, with resultant respiratory, circulatory, and nervous system disturbances. Partial obstruction, if allowed to persist, may cause an increase in blood pressure, tachycardia, a bounding pulse, or respiratory disturbances, such as gasping respirations, cyanosis, formation of mucus, or dyspnea. If obstruction is mainly inspiratory, pulmonary edema may be the result. There is also danger of pathologic changes in the central nervous system, especially in the cortical cells and vital centers. Noisy respiration is a sign of an obstructed airway and should not be tolerated. However, it must be noted that if respirations are depressed, there may be no noise. The anesthetist should have access to the patient's airway at all times, even during operations about the head and face, because it is not safe for the anesthetist to be unable to control the patient's respirations if it becomes necessary. The most important duty of the anesthetist is to maintain a free, unobstructed airway at all times.

Laryngospasm is a spasm of the adductor muscles of the vocal

cords and causes complete or partial obstruction of the airway. Partial spasm may be recognized by wheezing, crowing, or grunting, or other noises accompanying respirations. Symptoms of complete spasm are apnea and inability to inflate the thorax. The causes of laryngospasm are varied. A common cause is the presence of mucus, blood, or vomitus. A high concentration of the anesthetic agent, especially ether, will also produce the spasm, as will the presence of soda lime dust, especially when a to-and-fro filter is being used. Other causes include anoxia, mechanical stimulation, and trauma from airways, laryngoscopes, suction tips, or other foreign bodies. The production of laryngospasm is also possible from reflex stimulation, such as traction on the viscera or trachea, dilation of the rectum, or stimulation of the periosteum, vagina, or perineum. Parasympathetic stimulation and sympathetic depression from the use of drugs are other possibilities.

When the spasm is partial, the following measures may relieve it. The possibility of excessive carbon dioxide should be ruled out. Then the oxygen concentration of the inhaled mixture should be increased, or 100 per cent oxygen given under pressure. An artificial airway should be inserted and secretions aspirated from the pharynx. If the obstruction is not then relieved, an intratracheal catheter should be inserted and atropine sulfate given intravenously.

When laryngospasm is complete, the thorax should be inflated with oxygen, using bag and mask, intubation performed, and oxygen given.

Circulatory Complications

Circulatory complications during anesthesia are usually characterized by a change in the rate and character of the pulse, an elevated or depressed blood pressure, and changes in pulse pressure and are closely associated with respiratory and neurologic complications.

Tachycardia, a rapid pulse rate, may be caused by excitement during the induction period, loss of blood, or shock or trauma during the operation. The use of atropine as premedication is another cause, commoner in children than in adults, as is the use of sympathomimetic drugs in conjunction with anesthesia. Tachycardia may also be produced by the action of the anesthetic agent on the conducting tissues of the heart.

Bradycardia, a slow pulse rate, may be produced by stimulation of the carotid sinus reflex, increase in the irritability of the conducting tissues of the heart, or vagal stimulation. Bradycardia also occurs in respiratory failure accompanied by asphyxia or anoxemia.

During anesthesia, the types of arrhythmias (disturbances in the normal rhythm of the palpable pulse) that may occur vary from extrasystoles to ventricular tachycardia. In cases of cardiac disease, the effects of the anesthesia and the operation may produce arrhythmias. Under any circumstance, severe respiratory depression or deep anesthesia are

possible causes. The use of epinephrine during anesthesia has also been incriminated. When chloroform, cyclopropane, or ethyl chloride is the anesthetic agent, arrhythmias may result from the attendant increase in cardiac irritability. Another possible cause is vagal stimulation, such as attends stimulation of the carotid sinus, hilus of the lungs, aortic plexus, or trachea.

If the arrhythmia is due to deep anesthesia, the anesthesia should be lightened and the concentration of oxygen increased. The carbon dioxide absorber should also be checked to rule out the possibility of excessive carbon dioxide. If the condition is due to the anesthetic agent, another agent should be substituted for the one causing the arrhythmia. Novocain, 1,000 cc. of a 1 per cent solution, about 200-300 cc. given fairly rapidly, then at a rate of ten to twelve drops a minute, may prove beneficial. In most instances, arrhythmias during anesthesia may be detected by palpation of the pulse. However, electrocardiographic recordings may at times be necessary.

Hypotension with a decreased pulse pressure and tachycardia is usually the result of shock, overdosage of a depressant drug, deep anesthesia, or cardiac disease. Hypotension with a decreased pulse pressure and bradycardia usually accompanies deep anesthesia, reflex stimulation, or spinal anesthesia.

Hypertension with an increased pulse pressure and tachycardia may be due to anoxia, asphyxia, the use of epinephrine, thyrotoxicosis, or a prolonged excitement stage. When the hypertension is accompanied by increased pulse pressure and bradycardia, the cause may be increased intracranial pressure, anoxia with anesthesia, or the use of cyclopropane as the anesthetic agent.

In sudden cardiac arrest, the pulse is suddenly imperceptible. Respiration continues but soon ceases. An ashen gray cyanosis slowly appears. Heart sounds are imperceptible, the pupils are widely dilated, and the blood pressure cannot be obtained. Outstanding causes of sudden cardiac arrest are ventricular fibrillation, which may occur during the excitement stage or in the presence of thyrotoxicosis; reflex stimulation; stimulation of the vagus; hyperactivity of the carotid sinus; embolism, pulmonary or from air or fat embolus; cardiac failure due to coronary occlusion; a toxic anesthetic agent, especially chloroform, ethyl chloride, or a local anesthetic.

If the heart is exposed, the treatment of cardiac arrest includes cardiac massage, an injection of epinephrine (0.5 cc., 1:1,000 solution) into the right auricle, an injection of 2-4 cc. novocain (1 or 2 per cent), an injection of 50 cc. blood into the ventricles and massage, and the administration of oxygen and artificial respiration. If the heart is not exposed, epinephrine (0.5 cc., 1:1,000 solution) is injected into the right auricle, oxygen and artificial respiration are administered, and novocain (50-100 mg.) and adrenalin (1 cc.) are given intravenously. If no re-

sponse is evident, the chest should be opened at once without preparation and cardiac massage instituted. Adrenalin should *not* be given if cyclopropane is being used.

Neurologic Complications

Neurologic complications result from undue stimulation of the nervous system. Autonomic nervous system disturbances are manifested by changes in the circulatory and respiratory systems. Symptoms of neurologic disturbances are increased muscular activity, rigidity, and convulsions. The involuntary muscular contractions of convulsions during anesthesia result from increased irritability of cortical cells and other cells that control motor functions. Asphyxia or hypercapnia can cause convulsions, as can pressure on or stretching of nerves due to awkward positioning, overdosage of a local anesthetic when used in conjunction with a general anesthetic, stimulation of motor centers by such drugs as evipal and vinethene, and pre-existing changes in the nervous system that have no relation to the anesthesia. Convulsions may also be idiopathic, e.g., "ether" convulsions.

When convulsions are due to asphyxia, the asphyxia is obvious, and the convulsions are spasmodic, beginning in small muscles and gradually involving the larger muscles. The anoxemia should be immediately relieved by re-establishing the airway; if the convulsions persist, ultra-short-acting barbiturates should be given intravenously. Convulsions due to hypercapnia accompany rebreathing, or a high concentration of, carbon dioxide (15 per cent or more) and are characterized by twitching of small muscles with gradual involvement of larger muscles. Oxygen should be given, and rebreathing eliminated. The machine should be checked in order to confirm the identity of all gases and to make sure that the carbon dioxide is not on the wrong yoke.

The cause of idiopathic, or "ether," convulsions remains unknown. However, among the suspected causes are hypercapnia, alkalosis, hypocalcemia, disturbance of carbohydrate metabolism, liberation of a neurotoxin during anesthesia, impure air or oxygen, an overdose of atropine, and overoxygenation. The outstanding features of idiopathic convulsions are: (1) They are seen in youths and children more often than in adults and in the presence of septic infections and high fever. (2) They occur during deep anesthesia and are not relieved by lightening or deepening the anesthesia. (3) They usually occur during ether anesthesia, but they may occur during the use of other agents. (4) They are not confined to any part of the body but are generalized. (5) They usually develop after anesthesia has been established for twenty or thirty minutes, although they may occur earlier.

To control idiopathic convulsions, the administration of the anesthetic should be immediately discontinued and the inhaler discarded. A patent airway should be established and intuba-

tion performed if necessary. Ultra-short-acting barbiturates should be given slowly and in amounts sufficient to control the convulsions. Alkalinizing solutions should be given intravenously and oxygen given by catheter or semiclosed mask. If idiopathic convulsions are allowed to persist without treatment, death will occur from asphyxia and circulatory failure. If treated sufficiently early, the patient will recover.

When sweating occurs during anesthesia, the cause is usually excessive covering of the patient, oxygen lack due to obstruction of the airway, or the use of sympathetic nervous system stimulants.

TECHNICAL COMPLICATIONS

Technical complications arise from the use of defective apparatus or improper manipulation of equipment. The result may be an inability to maintain a constant level of anesthesia, hypercapnia, or anoxia. The difficulty can usually be traced to faulty machines and equipment; consequently, these should be kept in excellent condition at all times.

One should be constantly on the alert for explosion hazards, for accidents from explosions are among the most serious that may occur during anesthesia. Sources of ignition are flames, electrical equipment and switches, static electricity, and the use of oil on the anesthesia machine. All switches and electrical apparatus which are to be used in the operating room should be flameproof if possible. Switches should be of the mercury type; if this is not possible, they should be located outside the operating room. All switches, lights, and other electrical equipment should be grounded. The use of explosive agents should be avoided when electrical apparatus is being used. Static discharges are the most difficult to eliminate. Static electricity may be reduced by increasing the humidity of the operating room to above 55 per cent. The use of woolen blankets or of silk or rayon garments should be forbidden, and rubber-soled shoes should not be worn. Overcrowding of the operating room should be avoided, and visitors, doctors, and nurses should not be allowed to touch the anesthesia equipment.

Before the administration of an anesthetic is begun, the breathing tubes and bag should be wet. The face mask should not be removed more often than is absolutely necessary. The Horton intercoupler has been employed to keep the patient, the operating table, the anesthetist, and the anesthesia machine at the same potential. However, when the intercoupler is being used, it does not mean that all other precautions may be ignored.

SUMMARY

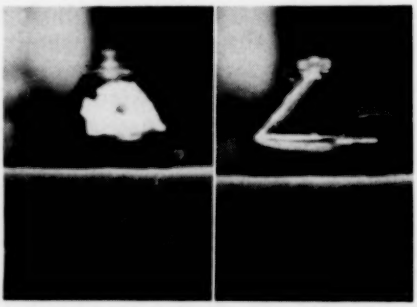
The respiratory, circulatory, neurologic, and technical complications that may occur during anesthesia have been described, and means for their control have been outlined.

The importance of keeping equipment in excellent condition has been stressed, as has the need for strict precautions in the prevention of explosions.

NOTES

When anesthetists get together, they talk about anesthesia. They talk about gadgets, special technics, and interesting cases. This section of NOTES was originated so that anesthetists could exchange ideas in writing as they do in conversation. Send in your contribution now. Other anesthetists will be helped by it.

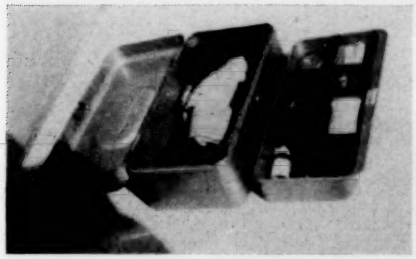
Infant's Ether Mask.—When I have a very small baby to anesthetize, I use a small tea strainer as an ether mask. The strainer may be obtained at any dime store. The handle is detachable and holds the gauze in place just like a real mask.—Janet D. Cabedo, R.N., Bronx, N.Y.



Mask for Use with a Jutte Tube.—The mask illustrated is airtight with ordinary bag pressures. A hole, 5/16 or 1/4 in. in diameter, is drilled in the metal shell. (Being righthanded, I find it convenient to have the hole in the portion of the mask at the right corner of the mouth.) A hole is cut in the rubber section of the mask directly under the hole in the metal shell. In the center of each of two 3 by 2 in. pieces of open penrose tubing or rubber glove is cut a 3/16 in. cross. With the cross-cut in slightly different directions, these two pieces are fitted over the outside of the rubber mask and firmly secured with rubber cement. This may or may not have to be reinforced with strips of adhesive. With the metal shell fitted over snugly, this is more satisfactory and airtight than an all rubber mask fixed the same way. The tip of a mosquito forceps is inserted through the cross-cut to catch and draw through the end of the stomach tube. The tube may then be connected to a suction apparatus or allowed to drain freely according to the surgeon's desires.—Harriet L. Aberg, R. N., Galesburg, Ill.

Anesthetist's Kit.—The anesthetist's kit used at Northwestern Hospital, Minneapolis, is an ordinary cashier's box. In the divided removable tray on top, the following necessary equipment is kept: stimulants and other ampule medications, blood pressure dial, tourniquet, ether corks, vinethene caps, safety pins, files, tongue blades, and

washed ace bandage for restraining an arm. In the bottom of the box are kept the sphygmomanometer, stethescope, suction ap-



paratus, and airways. Our staff members find the kit very compact, and the convenience of the arrangement makes any small piece of equipment readily accessible to the anesthetist.—ELEANOR OLSON, R.N., Minneapolis.

SUPPORT FOR LATERAL POSITION ON OPERATING TABLE.—To maintain patients in the lateral position on the operating table to the satisfaction of the surgeon and anesthetist and to the best possible advantage of the patient is difficult.

From the surgeon's viewpoint the operative field must be well exposed, room allowed for manipulation, and the position steadily maintained. The anesthetist is concerned with securing for the patient a free airway and respiratory excursions unhampered by the weight of the shoulder and upper arm, with such fixation as will prevent the patient from rolling either forward or backward, due care being exercised to prevent either strain or pressure from producing possible nerve injuries. The head should be pillowed so that cervical and dorsal spines are in alinement, the face should be turned slightly downward to facilitate drainage of secretions from the airway, and the blood vessels of both arms and hands should be accessible for the administration of intravenous therapy.

The effort to achieve these objectives led to the development of the device now to be described. The structural requirements of such a device are adjustability to various breadths of shoulders and sufficient rigidity to hold firmly the largest and heaviest patient. It should be freely movable along the siderails of the table. It should support the upper arm from shoulder to and including the hand.

The device shown in the illustration was constructed by the engineer at Lutheran Hospital, Fort Dodge, Iowa. If produced in a fully equipped factory a few worthwhile refinements could be made that are beyond the capacity of a hospital shop.

A piece of strap iron of a size to fit easily into the siderail slides of the operating tables was bent at right angles, the vertical ends being long enough to reach a little above the broadest shoulders and the horizontal bar being just long enough to reach across the table. The extension for the forearm is fastened to the under side of the horizontal bar by means of a small bolt fitted with a wing nut. Four holes near one end of the horizontal bar permit adjustment to the length of the upper arm.

The patient is allowed to assume the desired position before

being anesthetized so that he can co-operate in securing the most advantageous position. In our experience patients seem interested and reassured rather than frightened by the positioning. For kidney operations the first consideration is the position in relation to the kidney elevator. Then the shoulders are brought back against the vertical bar protected by a suitable pad. In the illustration all padding was omitted to show the device clearly. The stethoscope is applied a little above the elbow with the cuff of the sphygmomanometer over it. A muslin wrapper, a casing in the end of which is slipped over the bar of the support, is then wrapped around arm and support. The cuff serves as padding for the arm. If it is not used, a soft thick pad should be substituted. All wrappers should be placed on the middle portion of the arms, the regions near the shoulder and elbow joints where nerve injury might result being avoided. The support is adjusted about 1 inch above the arm so that the weight of the arm is borne by the support rather than by the patient's respiratory muscles. The forearm is supported similarly or on a basswood splint — whichever method best keeps the blood vessels accessible for intravenous injections.

Personal experimentation, we believe, is the best way of learning just where the support should be placed on the siderails. In most cases 5 to 7 inches below

the shoulder level seems about right. When the table is broken in the middle, the brace slants toward the patient's head; this permits the shoulder to encroach upon the neck and puts considerable stretch upon the upper chest wall, thereby impeding chest movement. Moving the brace a little farther down on the side-rails improves the position. Two radially grooved, opposing discs, such as are used on some knee crutches, placed in the vertical bars of the support, would facilitate finding the best position for a particular patient with a particular degree of angulation of the table. Here personal experience is the best teacher. Making the grooved discs is probably a factory job, as is the use of aluminum or magnesium to reduce the weight of the support.

We have found that reversing the Trendelenburg position as the table is broken, until the torso is horizontal, helps both respiration and circulation. Because the picture was taken from a high camera angle the head seems to be down. In fact, the torso was horizontal. The head down position plus the other factors that limit respiratory excursion places the patient in a position of mechanical disadvantage and is no advantage to the surgeon.

A 3 inch adhesive strap is placed over the region of the greater trochanter and fastened at both ends to the sides of the table to prevent the hips from rolling either forward or backward. The short curved brace furnished with all tables to keep the patient from rolling back may be used. However, with hips and shoulders fixed, it seems unnecessary, although it does no harm. We do not use the longer brace designed to keep the patient from rolling forward. We believe that its use greatly restricts abdominal respiratory movements which should be as free as circumstances will permit.

The under leg is flexed at the hip and knee joints and held in position by placing the knee strap over the leg just below the knee. The upper leg is extended, and its entire length is supported on a large pillow.

SUMMARY

A support to maintain a patient in the lateral position on the operating table has been described and illustrated. The objectives are to give as good exposure and as much freedom for manipulation as possible, while reducing the handicaps placed upon the patient's respiration and circulation by the inherently difficult position.—LOUISE SCHWARTING, R.N., Fort Dodge, Ia.

PHOTOGRAPH OF SPEAKERS' TABLE AT ANNUAL BANQUET AVAILABLE

Send request for print with ONE DOLLAR to American Association of Nurse Anesthetists 22 East Division St. Chicago 10, Ill.

LEGISLATION

DO ANESTHETISTS NEED LIABILITY INSURANCE?

Emanuel Hayt, LL.B.*

It is the impression of many nurse anesthetists that they are protected against claims for injuries to patients by the hospital's malpractice or liability insurance. Such is rarely the fact. A letter from a Colorado attorney, defending a damage action against a nurse anesthetist, states: "Unfortunately, the hospital which is a co-defendant in this case carried liability insurance which protects the hospital but not its employees, and unfortunately this insurance is of no benefit to my client unless the plaintiff is able to settle the matter with the insurance company and release her from any liability, which does not look likely at the moment. Because of this, my client is having to stand the expense of defending this action herself."

In that case, a paying patient was admitted to a voluntary hospital where, in due course, she was taken to the operating room. While she was still under the effects of the anesthetic the "defendants and each thereof," according to the complaint, "negligently caused a severe and damaging burn upon said plaintiff's left eye." Damages in the amount of twenty-five thousand ($25,000.00) were demanded. Both the anesthetist and the hospital were sued. The case is awaiting trial.

A number of other recent cases illustrate the type of anesthesia accidents which occur, resulting in serious injury to the patient and claims for damages against those alleged to be responsible.

The parents of a 9 year old girl in Washington brought an action to recover damages for her death, which occurred while she was under an anesthetic during an eye operation.

The child had been given the usual preoperative tests at the hospital and was taken into the operating room. The anesthetic, ether, was administered by the nurse anesthetist in the employ of the hospital. During the operation the surgeon and the nurse noticed that the child had become cyanotic. He immediately stopped the operation and passed to the other side of the drape to look at the child, who was not breathing. This was the first and only sign that anything abnormal was occurring. Efforts were immediately begun to resuscitate her. Other doctors were called in to assist, but all efforts were in vain.

An autopsy revealed that the death was caused by an allergy to ether of the thymus gland.[1]

An 8-month old child in California smothered while unconscious after a minor operation at a hospital. The parents alleged

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*Counsel for A.A.N.A.; Counsel for Greater New York Hospital Association; Recipient of *Modern Hospital* gold medal.

1. Yeager v. Dunnavan, 174 P. 2d 755 (Wash.).

negligence in the failure to watch and protect the child while unconscious and under the influence of a general anesthetic. Death was due to atelectasis.[2]

In New York a patient brought suit for a burn received on her back while in a charitable hospital. The burn was not on an area of the body upon which the operation was performed. There was no explanation given to the patient as to why the burn should have occurred in the operating room while the patient was under anesthesia.[3]

A patient entered a hospital to have a wart on her nose removed and her tonsils excised. She was completely anesthetized by a nurse anesthetist; the surgeon used an electric needle to remove the wart. As he was cauterizing the wound there was an explosion, described as a "flash" and a "pop," about six inches above the face and apparently within the oral and nasal passages of the unconscious patient.

It appears that the "flash" resulted from the electric needle igniting some combustible gas, either the nitrous oxide or the ether. The evidence showed four possible causes of the "flash" and explosion: the needle may have ignited (1) ether, or (2) nitrous oxide contaminated by unclean anesthetizing apparatus, or (3) nitrous oxide contaminated through an unidentified agent at some time after it was received from the manufacturer, or (4) nitrous oxide which was already contaminated at the time of purchase.[4]

2. Thomas v. Seaside Memorial Hospital, 183 P. 288 (Cal. App.).
3. Pliss v. 83rd Foundation, 69 N.Y.S. 2d 727.
4. Diermann v. Providence Hospital, et al., 179 P. 2d 603 (Cal.).

The possiblities of fatalities in anesthesia administration are well recognized, despite all known safeguards and precautions. Death may be caused by an overdose of the anesthetic agent, cardiac failure, hypoxia, respiratory failure, respiratory obstruction, cardiorespiratory asphyxia due to vomitus, atelectasis, ventricular fibrillation, hemorrhage or shock, or some patient's disease.

Factors contributing to anesthesia fatalities may be inadequate or excessive sedation, error in judgment, poor choice of anesthesia, excess dose of agent, respiratory tract obstruction during the operation, laryngospasm during operation, inadequate or poor therapy, error in technic, inadequate supervision, inefficient resuscitation, or respiratory obstruction in the postoperative period.[5]

There are few fields in which errors are more likely to be the cause of serious or perhaps fatal consequences than in the administration of anesthesia. The patient on the operating table is helpless to prevent harm to himself; he must depend entirely on the judgment and skill of those in the operating room. His life is in the hands of his guardians.

IMMUNITY OF THE HOSPITAL

It is ordinarily a principle of law that where an employee is negligent both the employer and the employee are liable in damages. The exception to the rule is in the case of voluntary hospitals which, by reason of their

5. Anesthesia Study Commission, J.A.M.A. 135: 881, Dec. 6, 1947.

exempt status, are usually held not responsible for injuries to patients. The nurse anesthetist, however, is always liable irrespective of whether or not the hospital enjoys immunity, provided, of course, that she has been guilty of negligence. The only states in which the hospital may be held jointly liable with the nurse anesthetist are Alabama, California, Florida, Minnesota, and Oklahoma.

An almost invariable condition of exemption in most states is that due care had been used in selecting the persons employed or acting as agents.[6] The reason that due care is required in the selection of employees is that carefully selected individuals are less likely to commit errors which will injure patients. Patients entering a hospital should anticipate skilful and humane treatment, with a feeling of confidence and security. If the hospital has been reasonably prudent in investigating the qualifications of its nurse anesthetist who was guilty of the wrongdoing, it is usually relieved of further responsibility.

The majority of the courts, although agreed upon exemption, are by no means in harmony upon the reason for the rule. Some courts use the trust funds doctrine, which holds that damages cannot be recovered from an eleemosynary institution, because charitable funds may not be diverted to other purposes. Other courts base immunity upon the theory that *respondeat superior*, which means let the superior respond, does not apply where the service is for the benefit of humanity and not for gain. Others ground it upon public policy, that, as the institution is engaged in work highly beneficial to the state and to humanity, its funds ought not to be applied to the payment of private claims for damages. And still others base it upon the theory of an implied agreement on the part of one accepting the benefits of a charity not to hold it liable for damages.

Many hospitals carry no public liability insurance, because charitable institutions in those states are not legally responsible for personal injuries to patients. Insurance is intended to protect one against loss or liability from some hazard.[7] Obviously, if no risk is involved for which damages may be paid, there is no reason for spending money for insurance premiums.

Another reason some hospitals do not have liability insurance is the fear that such coverage may create liability where none otherwise exists. With few exceptions, the courts have held that insurance will not of itself impose liability upon a charitable organization if no responsibility is provided by law. The rule is even more true of individuals: insurance of itself does not establish liability if none exists at law, and if the facts accepted by the court or jury indicate that the occurrence was not the result of negligence on the part of the individual.

Relationship to the Hospital

In excluding charitable hostals from liability, one of the legal concepts is that the nurse

6. Silva v. Providence Hospital of Oakland, 14 Cal. 2d 762, 97 P. 2d 798.

7. 14 R.C. L. 839.

anesthetist, in performing her duties in the operating room, does not act as a worker of the hospital: she is said to be carrying out the orders of the surgeon to whose authority she is subject. The hospital merely procures for the patient the services of the anesthetist; it does not undertake through the agency of the anesthetist to render those services itself.[8]

Another concept is that the doctor in the operating room has temporarily borrowed the anesthetist; that for the time being she is under his direction and control and not under that of the hospital, by whom she is in fact employed. Although in the general employ of the hospital, she is the immediate agent of the operating surgeon.[9]

RESPONSIBILITY OF THE SURGEON

Despite the fact that the anesthetist employed by the hospital ceases to be the agent of the hospital while she is in the operating room, the surgeon does not necessarily become responsible for her acts. It is true that her duties in the operating room are under the control of the surgeon, yet he is not liable for her negligence unless he had, or in the exercise of reasonable care should have had, knowledge of the careless act being performed by her.

The physician has the right to assume that the anesthetist employed by the hospital is competent. He is not chargeable with her incompetency unless he was aware of her lack of experience or skill. A duty rests upon him, however, to give proper instructions to the anesthetist except as to her ordinary duties.[10] During the course of the operation he must see that no preventable injury occurs to the patient.[11]

NEED FOR INSURANCE

No physician or nurse is immune from the danger of a patient's charging him or her with malpractice or negligence. Such claims or suits are, to be sure, often without merit and may be brought at the instigation of a disgruntled patient or as an attempt to evade paying bills for professional services.

Under the cloud of such an attack a professional person is likely to put up a fight which may be only partially successful if he is doing it singlehanded. The trouble, worry, and expense of properly vindicating himself or herself frequently leads the doctor or nurse, if not the insurance company as well, to consent to compromises and settlements out of court in order to escape notoriety.

Under these conditions it is but natural that the number of malpractice or negligence claims and suits should be on the increase. One compromise invites another. The seriousness of the situation is recognized and admitted by members of medical and dental societies who also realize the urgent need of co-operating among themselves, not only in defense of their individual reputations, but in defense of their profession.[12]

8. Schloendorff v. The Society of the New York Hospital, 211 N.Y. 125, 105 N. E. 92.
9. Randolph v. Oklahoma City General Hospital, 71 P. 2d 607 (Okla.)
10. Morrison v. Henke, 165 Wis. 166, 160 N. W. 173.
11. Aderhold v. Bishop, 940 Okla. 203, 221 P. 752.
12. Physicians', Surgeons' and Dentists' Group Plan Liability Policy. The Aetna-Izer No. 14, p. 3, Oct. 1919.

It is well to recall to mind the series of articles which appeared not long ago in various lay and professional publications to discredit the nurse anesthetist and to create alarm in the public mind over the dangers of anesthesia administration by any but medical experts. Any accident involving a nurse anesthetist may be seized upon to discredit nurse administration of anesthetics. Since nurse anesthetists, as well as other persons engaged in medical activities involving life and death, are in a vulnerable spot, they should unite in defense of both their individual and professional reputations.

A nurse anesthetist having to defend a law suit brought against her by a patient may have to pay a considerable sum of money to defend the claim. Irrespective of whether she wins or not, she may suffer loss in prestige and reputation that cannot be measured in dollars and cents. A judgment against the nurse may wipe out her savings or other assets and jeopardize her future earnings through a salary attachment. Insurance, if adequate in amount and scope of coverage, will not only pay damages but all the costs of defense, including the attendance of witnesses and experts.

Members of the medical profession are more conscious of these possibilities than are nurses. Every doctor with a reputation to defend usually carries professional liability insurance.[13] Every medical journal favors such insurance for doctors, and the premium is within easy reach of every physician who is earning a living. *The Journal of the American Medical Association* each week publishes a summary of the facts and the decisions of the courts in cases involving physicians sued for malpractice or concerned in other litigation. Your own JOURNAL is now reporting legal matters affecting nurse anesthetists.

One reading the daily papers often see news items of doctors and other professional persons who are being subjected to lawsuits. These stories destroy many a professional reputation. Any accident or report of a legal action having a hospital, a physician, a nurse, or a patient as a topic of discussion or comment appeals to the public interest and readily receives publicity. The human interest angle is too strong to neglect this type of news story. The actual truth eventually may show that the professional person was not at fault at all, but that aspect is not determined until there has been an adjudication in court. By that time the incident has lost its news value; the result is ignored. The original inference with its consequent damage to professional standing remains unrefuted to public knowledge.

Professional insurance assures prompt and thorough investigation of the anesthesia accident by the insurance company investigators. The investigation may bring facts to the attention of the daily press which can possibly alter the presentation of the news report and give a fairer picture of the happening.

13. Professional Liability Insurance. Casualty, Surety and Miscellaneous Insurance Monthly Bulletin p. 27, May 9, 1929.

An Insurance Program for Anesthetists

There are many ways in which an insurance program for nurse anesthetists can be handled. If the policy is issued to individual members, certain state regulations will have to be complied with, resulting in a variety of contracts. The cost of issuing separate policies will be reflected in the premium. On the other hand, if a policy is issued on behalf of the members of the Association and a certificate given to each member in good standing, there would be a uniform contract involving considerably less clerical work for the insurance company.

The premium could be collected by the Association as part of the annual membership dues and then remitted to the company. Renewal of the policy would be automatic for old members; new members would become insured upon payment of dues. The contract can be issued on a one, three, or five year basis with a reduced premium for so doing. The greater the percentage of members who insure in a group policy, the lower will be the premium rate and the more effective the co-operation within the Association itself.

From motives of self interest alone, the company will spare no pains or expense in defending a claim or suit brought against any member insured under its group policy. Were it to do otherwise, the member affected would naturally make her dissatisfaction known to the other members who might be led to cancel their insurance.

The medical and dental professions have two types of policies issued to cover Physicians', Surgeons', and Dentists' liability: (1) individual and (2) group. These policies are offered to members of recognized medical and dental societies. The individual policy is used in a territory where no group policy is in force; this form serves as a nucleus for a group policy.

The basic limits under all forms of malpractice policies are $5,000 and $15,000, that is, the company's liability in one case is limited to $5,000 and, subject to the same limit, the company's total liability on account of all claims and suits arising during any one annual premium is $15,000.

A policy for anesthetists should provide that the insurance company agrees to pay all loss up to a specified amount, usually $5,000, by reason of liability imposed by law upon the anesthetist for damages on account of professional services rendered or which should have been rendered by her, including the administration of drugs, anesthetics, or medicine, or any counterclaims in a suit brought by the anesthetist to collect fees.

Only one out of six large insurance companies approached has been willing to write a policy for nurse anesthetists. This company specializes in insurance for hospitals and physicians. The company agrees to defend without charge every suit for alleged negligence, error, or mistake that may be brought against the nurse anesthetist arising out of the practice of her profession, irre-

spective of where she administers anesthesia, and regardless of whether she does it on a fee or salary basis, provided, of course, her act is performed under the direction or supervision of a licensed physician.

The company will settle no suit without the written consent of the assured; it agrees to carry every suit to the highest court to which an appeal can be taken when the lower court decides the case against the anesthetist.

The company also agrees to defend without limit of cost any suit filed at any time, and to furnish any bond not exceeding in amount the minimum limit of the policy, which may be incidentally necessary for such suits, for appeals or the release of attachments or garnishments.

At the present time the cost of a policy for 5/15 limits on an annual basis will be $20, and possibly $50 for a three year policy. After a sufficient time has elapsed, proper credit may be given for experience and eventually to reduce the cost. There is a strong likelihood that a group policy can be obtained for a 10/30 limit for an annual premium of $12. At the outset, however, the policy is to be issued on an individual voluntary basis.

Insurance companies are reluctant for some incomprehensible reason to insure nurse anesthetists against liability. One insurance broker in a recent letter writes: "For a period of some months, I have been working on the subject of securing Professional Liability Insurance for the members of the Tennessee Association. Thus far, most of the major companies have declined the risk because of its indefinite character and because of the resulting difficulty of arriving at a fair rate. I would very much appreciate it if you would pass on to me such information as you have secured with respect to this subject."

Obligations of the Insured Anesthetist

The anesthetist, upon the occurrence of an accident, is required to give immediate written notice thereof, with the fullest information obtainable at the time, to the home office of the company or to its duly authorized agent. The anesthetist is to give like notice, with full particulars, of any claim or suit made or brought on account of such accident and to forward to the company or its authorized agent all documents received relating thereto.

The anesthetist may not, without the written consent of the company, admit liability nor make any arrangement, offer, promise, or payment. Whenever possible, the anesthetist shall give all such information and assistance as the company may require. She must use diligence to prevent or minimize a claim being made under the policy.

The company will not be liable for any claim arising out of the anesthetist's liability under any Workmen's Compensation Act and/or Employers' Liability Law.

The insolvency or bankruptcy of the anesthetist will not release

(*Continued on page 342*)

THE NEWS

HOUSE OF DELEGATES REFERRED BACK TO COMMITTEE

Trust Fund Converted to Educational Fund

The revision to the bylaws proposing a House of Delegates was referred back to committee at the business session of the fifteenth annual meeting in Atlantic City. The majority of the members on a rising vote favored postponing action on the revision in order that the state associations might have more time to study the proposal.

Also referred back to committee was the revision on Junior Membership, the measure failing to gain approval because of disagreement about the wording.

The Association's Trust Fund was abolished, the money in the fund being transferred to a newly created Educational Fund. The resolution on the Educational Fund as accepted provides for the financial aid of graduate anesthetists who wish to prepare themselves to teach in a school of anesthesia (see page 334).

A revision making mandatory a one year course of training for candidates for the qualifying examination was approved. The bylaw becomes effective in 1951.

Mrs. Van Arsdale Elected President

Mrs. Myra Van Arsdale, chief nurse anesthetist, St. John's Hospital, Cleveland, was unanimously elected to the office of president at the annual business session. Mrs. Van Arsdale has served as chairman of the Credentials and Trust Fund Committees, as a member of the Board of Trustees, and as 1st vice president for two terms. Miss Mabel Courtney was elected 1st vice president; Miss Ruth Bergman, 2nd vice president; Mrs. Gertrude L. Fife, treasurer. Mrs. Julia Baines, Mrs. Josephine Bunch, and Miss Minnie V. Haas were elected to the Board of Trustees.

Gift to Educational Fund

A gift of $200.00 to the Association's Educational Fund from Miss Elizabeth Navarro, 4 E. Sixth St., New York City, was announced at the banquet, the money to be used as an outright grant. Also acknowledged were contributions of $200.00 each from the Tennessee and Missouri associations.

Second Award of Appreciation

Dr. George Crile was the recipient posthumously of the Association's Second Award of Appreciation at the fifteenth annual banquet. In presenting the award, Miss Lucy E. Richards, the president, said:

Top left: Miss Lucy Richards receiving from Mrs. Dorothy N. Ball a citation from the state associations. Top right: Miss Richards and Mr. James Hamilton, banquet speaker. Bottom (seated): Mrs. Gertrude L. Fife, treasurer; Mrs. Myra VanArsdale, president. (Standing): Mrs. Julia Baines, Miss Minnie V. Haas, trustees; Miss Ruth Bergman, 2nd vice president; Miss Mabel Courtney, 1st vice president; Mrs. Josephine Bunch, trustee. Trustees completing their term of office in 1948-49 are Mrs. Gertrude Troster, Maj. Edith Aynes, and Miss Verna E. Bean.

Thirty-seven years ago, a world-famous Cleveland surgeon made it possible for nurses to become the right kind of anesthetists. In 1911, at his instigation, the first formal school for the training of nurses in anesthesia was opened at the old Lakeside Hospital in Cleveland. The significance of his pioneer achievement is thrown into bold relief when we compare the caliber of the anesthesia service in American hospitals before and since that time. Living memorials to him are the 54 formal schools for the training of nurse anesthetists in registered hospitals in this country. Because of his vision and his confidence, the anesthesia service in over 75 per cent of the nation's registered hospitals is being conducted by nurse anesthetists educated in schools patterned after the one of which he was the founder.

Tonight, it is my pleasure, acting in behalf of the Board of Trustees and the members of the American Association of Nurse Anesthetists, to announce the Second Award of Appreciation in the honor and the memory of Dr. George Crile and in recognition of his contribution to the advancement of the nurse anesthetist.

Miss Richards then read a letter from the late Mrs. Crile who had been invited to receive the award in Dr. Crile's name.

I think I never have had an invitation give me greater pleasure than your from the nurse anesthetists, nor can I imagine any citation in recognition of Dr. Crile's interest and support that would mean more for me to receive, as he believed in the nurse anesthetist, and their distinguished work was always his pride.

Miss Richards Receives Citation

In appreciation of Miss Lucy Richards' conduct of the office of the president, the state associations, through their representatives at the annual meeting, presented her with a bouquet of red roses at the annual banquet.

As spokesman for the state associations, Mrs. Dorothy N. Ball, president of the New Jersey association, said:

We, as representatives of various states, wish to take this occasion to make a public statement of our appreciation to you, Miss Richards, and present to you these flowers as a token of our appreciation for the magnificent manner in which you have guided us for the past two years, during which our organization has experienced many difficulties.

INSTITUTE IN NEW YORK CITY

An Institute for Nurse Anesthetists, conducted co-operatively by the American Hospital Association and the American Association of Nurse Anesthetists, will be held at the Hotel Commodore, New York City, February 7-11, 1949.

Lectures and discussions of subjects of pertinent interest to the nurse anesthetist make up the program. There will be three morning sessions devoted to anesthesia clinics in various hospitals. There will be a round table discussion on selected drugs. One session will be devoted to the principles of departmental organization and management.

Applicants must be members of the American Association of Nurse Anesthetists, the American Hospital Association, or their institutions must be members of the American Hospital Association. The tuition fee is $25. Certificates will be awarded. Enrolment is limited to 125.

Application blanks may be had upon request to Dr. Charles T. Dolezal, Secretary, Council on Professional Practice, American Hospital Association, 18 E. Division St., Chicago 10, Ill.

HIGHLIGHTS OF EDUCATIONAL FUND RESOLUTION

Recognizing the need for qualified nurse anesthetist instructors in schools of anesthesia and the importance to the profession of qualified instructor personnel, the Board of Trustees appointed a Scholarship Fund Committee, with Mrs. Gertrude Troster as chairman, to prepare a resolution providing for the establishment of an Educational Fund. The highlights of this resolution as accepted at the annual meeting are:

. . . Whereas, it is the desire and the aim of the American Association of Nurse Anesthetists to aid and encourage anesthetists in securing training to qualify as instructors,

Be It Resolved: that the American Association of Nurse Anesthetists establish an Educational Fund and plan as follows:

I Name. This fund shall be known as the "Agatha Hodgins Educational Fund."

II Purpose. This "Fund" is designed to extend financial assistance to graduate nurse anesthetists in obtaining further education and training to become qualified instructors, or to instructors who desire and need additional training to become better qualified.

*IV Sources. Transfer of funds totaling $700.00 now in the Scholarship Fund, to be augmented by:

(a) Voluntary contributions or grants by state or local associations, or by individuals or groups of individuals.

(b) Memorial contributions, which shall be acknowledged by the Chairman of the Board of Trustees to members of the family of memorialized deceased as directed by donor.

V Applications. Applicants for awards shall be (a) graduates of a school of anesthesia whose graduates are permitted to take the qualifying examination for membership in the American Association of Nurse Anesthetists, and (b) shall be willing to pledge, and upon selection shall pledge, at least five years to the profession of instructing in a school of anesthesia, as may be required by said Committee.

VIII Amount and Repayment of Loan. (a) The amount that may be awarded to any one applicant shall not exceed $500.00, unless in special instances this amount may be increased by authorization of the Board of Trustees of the American Association of Nurse Anesthetists. . . .

(b) When application for loan has been authorized, the Committee, in its discretion, may require applicant to sign a negotiable promissory note, with one or more endorsers. Any such note shall be payable to the American Association of Nurse Anesthetists and provide for payment in installments at such dates and upon such terms as the Committee may require.

And Be It Further Resolved, at such time as the funds in the award shall warrant, the monies may be given as a grant as well as a loan and that such monies as are donated with such restriction shall be reserved for grants only. . . .

*A resolution abolishing the Trust Fund and transferring the money in that fund to the Educational Fund was approved at the fifteenth Annual Meeting.

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ABSTRACTS

ENGBAECK, LISE: Investigations on the course and localisation of magnesium anesthesia: a comparison with ether anesthesia. Acta Pharmacol. et Toxicol. **4**: Supp. 1,1-189, 1948.

"The anesthetic effect of magnesium was demonstrated in 1869 by Jolyet and Cahours. . . . During the last few years the use of magnesium combined with other anesthetics has become of greater interest, as the introduction of curare to induce more complete muscular relaxation during anesthesia (Griffith and Johnson 1942, Cullen 1943) may result in search for other substances with a point of attack which is at any rate partly peripheral, similar to that of curare, but the dosage of which is more easily determined, and for which an effective antidote exists in case of overdosing. Magnesium meets these demands, but it appears from earlier clinical publications that in man the margin between the effective concentration and that which produces respiratory paralysis is very narrow. In spite of the fact that numerous investigations have been made of various problems in connection with magnesium anesthesia, very little material has yet been published which gives a closer investigation of the anesthetic by means of quantitative reflex determinations, in relation to the concentration of magnesium in the blood. The purpose of the present work has principally been to make an analysis of the course of magnesium anesthesia by means of a quantitative determination of the reflex irritability simultaneously with a determination of the magnesium concentration in the blood, and a comparison with ether anesthesia, as ether is the aliphatic anesthetic most closely investigated. Furthermore, the author's object has been to determine the point of attack of magnesium, firstly the relation between the central and the peripheral effect, secondly the more exact localisation of the peripheral effect of magnesium in motor nerve, end plate and muscular substance . . . . In low concentrations magnesium lowers the blood pressure considerably. At higher concentrations it inhibits intra-cardiac conduction. The reducing effect on the blood pressure is—at least partly—due to a peripheral action . . .

"It has not been determined, according to existing literature whether the ether concentrations obtained during general ether anesthesia produce a peripheral effect on the motor system, i.e. the motor nerves, end plates, and muscles. The purpose of the experiment described in the present paper is the investigation of a possible peripheral effect of ether. If such an effect can be demonstrated it is the aim to investigate to what extent the ether effect on the extensor reflex can be attributed to a central origin. . . . The experiments were performed on rabbits. . . . The present work deals only with changes in reflex irritability since the observations were made during the course of the

anesthesia. . . . As this work deals with the course of rather short ether anesthesia, mixed venous blood was used for the analyses. . . .

"The experiments clearly show a connection between the ether concentration in the blood and the irritability of the cornea, the latter decreasing as the ether concentration increases. But as the slope and level of the curves vary considerably in the different experiments the material is unsuitable for drawing a general concentration-action curve. . . . In order to investigate the relation between the central and the peripheral effect of ether, a Ludwig electrode was placed at both the central and the peripheral end of the severed sciatic nerve, which made it possible to observe simultaneously the threshold of indirect stimulation and of the crossed extensor reflex. The normal threshold was determined as the value given by at least two successive identical threshold values, after the brief anesthesia required for placing the electrode and cannula in position was discontinued. Eleven experiments were made. . . . The dosage of ether was empirically determined, but the concentration in the blood was controlled for comparison with other experiments. . . . The experiments indicate that ether concentration inducing deep anesthesia and respiratory failure has a slight peripheral effect increasing the indirect stimulation threshold from 50 to 150 per cent. As all the animals died from respiratory paralysis and the respiration was impaired some time before death, the peripheral effect demonstrated may be caused by asphyxia and not, primarily, by ether. In order to eliminate this possibility seven further experiments were performed under ether anesthesia and artificial respiration. In three experiments an increase of the indirect stimulation threshold varying from 90% to 210% was found at cardiac arrest. In the remaining four experiments there was no increase or only a slight increase of the threshold. . . . The increase of the peripheral stimulation threshold occurred almost simultaneously with, or shortly before, the extensor reflex was abolished at maximum stimulation. In the first two of the four negative experiments the inspired ether concentration was fairly high, paralysis of the heart occurring rapidly (in 42 and 27 minutes respectively), while in other experiments concentrations were used which did not cause cardiac arrest before 60 minutes had elapsed. The lack of equilibration between blood and tissues cannot, therefore, explain the absence of a peripheral effect in these experiments. Asphyxia occurred with beginning impairment of the heart, although artificial respiration was applied, and the effect observed may well be due to the asphyxia. But when the threshold was increased, the increase occurred before respiratory arrest, at a time when the cardiac action presumably was not seriously affected.

"The irregular peripheral effect during universal ether anesthesia is presumably due to the fact that the concentrations obtained are near to the threshold of the

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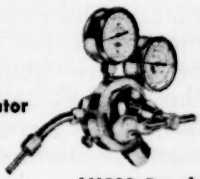

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peripheral effect. But the threshold increase of 150-210% is so great that it may scarcely be due to random variations of the threshold during the experiment. In the period before the experiment the threshold generally showed a slight decrease before becoming constant after preliminary ether anesthesia, this also demonstrating a peripheral ether effect. The fact that some of the former investigations have not demonstrated the peripheral effect of ether may be due to insufficient accuracy of the methods used for measuring changes in irritability. In order to investigate the relation between the central and the peripheral effect of ether it is necessary to compare the changes of irritability by indirect stimulation, and by elicitation of the extensor reflex. At both determinations changes in the peripheral irritability will be evident and may be presumed to run parallel, while changes in the irritability of the central nervous system will only affect the threshold of the extensor reflex. The effect on the extensor reflex may also partly be due to paralysis of the sensitive nerves, a possibility which cannot be excluded with the procedure used in the present investigations. Since, however, the motor nerve remains completely unaffected by ether in anesthetic concentrations, this possibility appears to be rather improbable. . . . As found with the corneal reflex, there is a clear relation between rising ether concentrations in the blood and in increase of the threshold stimulus of the extensor reflex, but the corneal reflex is affected at considerably lower concentrations than the extensor reflex. . . . For a more detailed study of the anesthetic stage the ether concentration has been determined at which the conjunctival, the pinch reflex, and the reaction to skin incision were abolished. The pinch reflex was abolished shortly before the conjunctival reflex. . . .

"The reaction to skin incision was examined three times and was found to be abolished almost simultaneously with the pinch reflex. At the concentration of 1.0 per mille which abolished the conjunctival reflex, the threshold stimulus for the extensor reflex was increased 2-5.5 times. The condition where the pinch and the conjunctival reflexes are abolished, and the threshold of the extensor reflex has been increased 2-5 times, may be termed deep surgical anesthesia as at this stage it is possible to perform major operations with no reaction from the animal. . . . In rabbits deep surgical anesthesia occurred at ether concentrations of 0.9-1.1 per mille in mixed venous blood. It occurred when 4.4 vol. % ether had been inhaled, the conjunctival and the pinch reflexes being abolished, and the extensor reflex after 51 and 72 minutes respectively reaching a level 2-3 times as high as the initial value, and this level was kept during the remaining part of the experiment (20 and 29 minutes). At lower concentrations (2.9-3.0 vol%) the conjunctival and the pinch reflexes were abolished, whereas at higher concentrations (4.8-5.0 vol%) the extensor reflex was absent after

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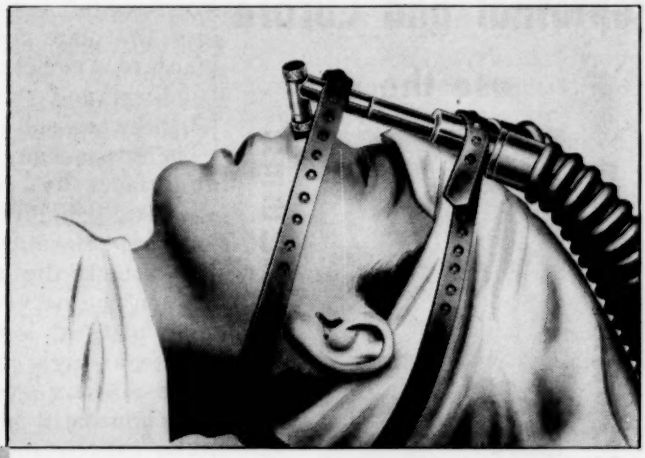
70 and 83 minutes respectively. . . . The corneal reflex and the plasma magnesium concentration have been examined in 17 experiments, where the effects of magnesium, given subcutaneously, on the corneal reflex and on the postural reflexes have been compared; and in a further 12 experiments, the effects of intravenous magnesium on the corneal and extensor reflexes have also been compared. The individual experiment shows a clear relation between a rising magnesium concentration in plasma and an increase of the threshold stimulus for the corneal reflex; a very slight change in the magnesium concentration may have a considerable effect on the corneal irritability. . . . The postural stages were affected earlier and abolished later than the corneal reflex.

"The individual experiment shows that the corneal reflex irritability decreases with a rising magnesium concentration plasma, but the data were not suitable for determining a complete concentration-action curve. . . . A variation of a similar order of magnitude is found when the corneal reflex irritability is related to the ratio magnesium concentration/calcium concentration in plasma. . . . In five experiments without artificial respiration an increase of 200-800% in the threshold of indirect stimulation was found at respiratory collapse. . . . Thus the indirect but not the central irritability is reduced at respiratory paralysis. . . . The results are explained as a reduced irritability of the reflex centre when the magnesium anesthesia was continued with rising concentrations after respiration had ceased. At the start of most of the experiments it was found that the indirect irritability decreased while the threshold of the extensor reflex either remained constant or decreased. . . .

"The crossed extensor reflex and the magnesium concentration in plasma were examined in 14 experiments. There was a clear connection between rising magnesium concentration in plasma and an increase of the threshold stimulus for the extensor reflex. . . . A comparison between the time when the various reflexes were affected during magnesium and ether anesthesia showed rather big differences. During ether anesthesia the postural and corneal reflexes were affected simultaneously and at considerably lower concentrations than the pinch and conjunctival reflexes. During magnesium anesthesia the corneal reflex was abolished later than the postural and pinch reflexes, the conjunctival reflex simultaneously with the extensor reflex, while—under ether—the conjunctival reflex disappeared before the extensor reflex. This sequence of abolition of the reflexes indicates a peripheral point of attack of magnesium anesthesia.

"The results of previous investigations of the more exact localisation of the pheripheral effect of magnesium indicate that its site is the end plate, while the question of a possible magnesium effect on the muscular substance

"FOR THOSE WHO HAVE NEVER LEARNED TO GIVE ETHER OR WHO HAVE FORGOTTEN HOW— INTUBATION IS FREQUENTLY A HAZARDOUS ADVENTURE . ."



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*Flagg, P. J.: The fenestration operation: major hazard — the anesthetic, (editorial), Am. J. Surg., 72: 497-499 (Oct.) 1946

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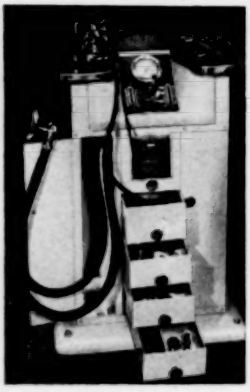

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has been less closely examined. The effect on the motor nerves does not set in until 10-20 times the concentration required for a magnesium block. Earlier investigations indicate that the motor end plate contains two interfaces: 1) between the nerve endings and the end plate, and 2) between end plate and muscle. The magnesium effect on these interfaces has not been studied before. . . . curarine, quinine-methyl-chloride, and magnesium all attack the transmission between the nerve endings and the end plate at low concentrations, while at high concentrations the transmission between end plate and muscle is also attacked. . . . The experiments indicate that—like curarine—magnesium does not prevent the liberation of acetylcholine but, somehow, prevents acetylcholine from acting on the end plate."

LEGISLATION

(*Continued from page* 330)

the company from payment of damages sustained or loss occasioned during the life of the policy.

The policy may be cancelled at any time at the request of the anesthetist, in which case the company shall, upon demand and surrender of the policy, refund the excess of the paid premium above the customary short rate premium for the expired term. By giving five days' notice the company may cancel the policy for the remaining period.

BOOK REVIEWS

A MANUAL OF PHARMACOLOGY. By Torald Sollman, M.D., Professor Emeritus of Pharmacology and Materia Medica, School of Medicine, Western Reserve University, Cleveland. ed. 7. Cloth, 1132 pages. Philadelphia: W. B. Saunders Co., 1948.

This seventh edition brings up to date the information on and concepts of drug actions. Many of the new drugs are included without excluding the older drugs of real importance. The information is presented in outline form and is supplemented by references to writings from which greater detail may be obtained. An extensive bibliography and an index complete the volume.

THE HUMAN BODY AND ITS FUNCTIONS: AN ELEMENTARY TEXTBOOK OF PHYSIOLOGY. By C. H. Best, C.B.E., M.A., M.D., D.Sc. (Lond.), F.R.S., F.R.C.P. (Canada) Professor and Head of Department of Physiology, Director of the Banting-Best Department of Medical Research, University of Toronto; and N. B. Taylor, V.D., M.D., F.R.S. (Canada), M.R.C.S. (Eng.), L.R.C.P. (Lond.), F.R.C.S. (Edin.), F.R.C.P. (Canada) Professor of Physiology, University of Toronto. Cloth. 500 pages. Illustrated. New York: Henry Holt and Co., 1948. $3.60.

This is an expanded and largely rewritten version of the original text. The subjects of the structure and function of the tissues and organs of the body are presented in a less elementary manner than was used in the first edition. The simplified terminology and statements of scientific facts have been retained. Of particular value to students of anesthesia are the excellent presentations of the subjects of blood, circulation, and respiration, as well as those concerning the nervous system.

TEXTBOOK OF PHARMACOLOGY AND THERAPEUTICS. By Harold N. Wright, M.S., Ph.D., Associate Professor of Pharmacology, University of Minnesota; and Mildred Montag, R.N., M.A., Director, Adelphi College School of Nursing, Garden City, N.Y. ed. 4. Cloth. 720 pages, 95 illustrations. Philadelphia: W. B. Saunders Co., 1948.

This book is revised to include the recent rapid advances in pharmacology and therapeutics. Each chapter is followed by a bibliography and a list of questions for use by students. Sources, types of drug preparations, systems of weights and measures, including calculation of dosage, solutions, prescription reading, and administration of drugs are only a few of the subjects presented in various chapters.

Discussion of the fundamental types of pharmacologic action of drugs and of the classification of drugs precedes the presentation of the drugs themselves. Each major division of the chapters pertaining to the drugs is based on their pharmacologic actions.

Of particular interest to students of anesthesia will be chap-

ters XI to XVII inclusive. The drugs which affect respiration and the nervous system, including narcotics, stimulants, and anesthetics, as well as the therapeutic gases, are discussed in these chapters. Local anesthetics are presented in chapter XX. In chapter XXI the drugs acting on the nerve endings of the autonomic nervous system or directly on the smooth muscle are presented. Chapter XXII presents a discussion of acid base, water and mineral balances and parenteral therapy. Drugs acting on the circulatory system and a discussion of blood are presented in chapters XXIII and XXIV. A glossary and 79 page index complete the volume.

Introduction to Human Physiology. By William D. Zoethout, Ph.D., Professor Emeritus of Physiology of Chicago College of Dental Surgery (Loyola University). Cloth. 424 pages, 138 illustrations, 4 color plates. St. Louis: C. V. Mosby Co., 1948.

This book was written for "the reader desirous of obtaining a fundamental knowledge of the operation of the human body but having no previous knowledge of the basic sciences of Physics and Chemistry." Because of this approach it may be useful as a preliminary review for students in courses of anesthesia. Index.

Physiological Chemistry. By J. F. McClendon, Ph.D., Research Professor of Physiology, Hahnemann Medical College, Philadelphia, Pennsylvania; Formerly Professor of Physiological Chemistry, Medical School, University of Minnesota, Minneapolis. ed. 7. Cloth. 463 pages, 83 illustrations. St. Louis: C. V. Mosby Co., 1946.

A general textbook on physiologic chemistry is an essential in the library of schools of anesthesia. A review of the general subject is often desirable, if not actually necessary before the more advanced study in courses of anesthesia. The book is divided into two parts, the first being a general discussion of the subject and the second being devoted to the laboratory work covering the field of the text. A list of international atomic weights, a glossary, a list of references, and an index complete the volume.

Observation and Study Guide for Student Teachers. By Edgar M. Cook, Ph.D., Professor of Psychology and Education, Carson-Newman College, Jefferson City, Tenn.; and John H. Cates, M.A., Principal of Jefferson City Elementary School, Director of Practical Teaching, Carson-Newman College. ed. 2. Cloth. 167 pages. St. Louis: C. V. Mosby Co., 1947.

A loose-leaf plastic bound book containing 30 work units. Each work unit is followed by a list of references pertinent to the particular subject. This handbook should be useful to nurse anesthetists who are entering the teaching field in schools of anesthesia.

Principles Governing Eye Operating Room Procedures. By Emma I. Clevenger, R.N., Supervisor, Eye Operating Room, New York Eye and Ear Infirmary,

Putnam

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BIBLIOGRAPHY • INDEX

G. P. Putnam's Sons • **2 West 45th St., N. Y. 19**

New York City. Cloth. 215 pages, 33 illustrations. St. Louis: C. V. Mosby Co., 1948.

This book was written as a reference book for nurses and physicians who are interested in planning and maintaining "eye operating rooms." The text is written in outline form and covers all of the phases of eye operating room procedures, from the prerequisites of the personnel to the list of manufacturers of instruments. The information presented in the book has been gathered from the experiences at the New York Eye and Ear Infirmary.

A short list of the anesthetics, general and local, is included in chapter 8, "Drugs and Solutions Used in Eye Operating Room Enumerated." In subdivisions of the chapter one page is devoted to the technics of administering the general anesthetics, avertin, ether, evipal, gas-oxygen, and pentothal sodium. Another page is devoted to the local anesthetics, giving the method and percentages of various drugs and solutions.

Private Enterprise or Government in Medicine. By Louis Hopewell Bauer, M.D., Diplomate, American Board of Internal Medicine; Member, Board of Trustees, American Medical Association; President, Medical Society of the State of New York. Cloth. 210 pages, 2 graphs. Springfield, Ill.: Charles C Thomas, Publisher, 1946. $5.00.

All persons who work in the hospital field should know about the arguments for and against government control, or so-called socialized medicine. This book presents the problems and answers the questions pertaining to this vital subject. The legislative proposals, the functioning of socialized systems of medicine in other countries, and the activities of private enterprise and of the American Medical Association are among the important phases of the problem discussed by the author.

Physiologic Therapy in Respiratory Diseases. By Alvan L. Barach, M.D., Associate Professor of Clinical Medicine, Columbia College of Physicians and Surgeons; Assistant Attending Physician, Presbyterian Hospital, New York City. Cloth. 408 pages, 74 illustrations. Philadelphia: J. B. Lippincott Co., 1948. $9.00.

This volume is an expanded version of the first edition, published under the title *Principles and Practices of Inhalation Therapy* in 1944. Many of the features of the first edition are retained, but in addition measures other than the therapeutic use of gases are described. Much of the text is unchanged, although certain chapters are rearranged and correlated. A brief introductory chapter gives the historical background of physiologic therapy in diseases of the respiratory tract. Anoxia is the main subject of two chapters. Pneumonia, pulmonary edema, war gas poisoning and lung irritants, and bronchial asthma are each discussed, with the physiologic bases and the important objectives in their treatment. The same pattern of presentation is used in the chapters on obstructive lesions of the respiratory tract, pulmonary

emphysema, bronchiectasis, pulmonary embolism and infarction, massive collapse of the lungs, pulmonary congestion, and congestive heart failure.

Accidental asphyxia and resuscitation, asphyxial problems of the newborn, and paralysis of the respiratory musculature are each discussed. In chapter 18 the specific problem of anesthesia and anoxia is discussed.

Methods of physiologic therapy, apparatus for inhalation therapy and the use of pressure, and the administration of aerosols are presented in a long chapter. Care of equipment, a chapter on oxygen analyzers, and a method of measuring gas concentrations complete the text. Each chapter or major division is followed by a bibliography.

ANESTHESIA: PRINCIPLES AND PRACTICES. By Alice M. Hunt, R.N., Associate Professor of Anesthesia Emeritus, Yale School of Medicine, New Haven, Conn. Cloth. Illustrated. New York: G. P. Putnam's Sons., 1948. $2.60.

This book is written for the nursing profession by a nurse anesthetist with many years of experience. The author has presented the results of her experience and her knowledge of the subject of anesthesia in a manner both concise and practical. Throughout the text emphasis is placed on the patient as the dominant factor, a point often overlooked in presentations on the subject of anesthesia.

The years during which the author has worked in the field of anesthesia have covered an era of great progress. This progress is apparent in this account of the author's activities.

Bibliographic footnotes, a biographic table, and an index complete the text.

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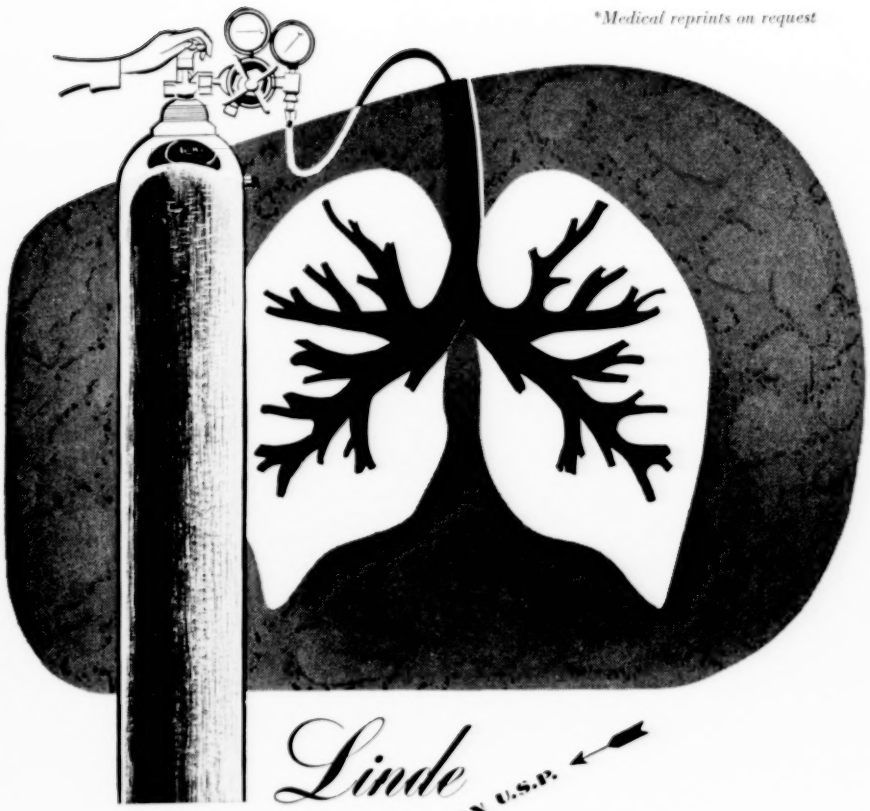

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